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BULLETIN

OF

THE JOHNS HOPKINS HOSPITAL

Entered as Second-Class Matter at the Baltimore, Maryland, Postoffice.

Vol. XXII.—No. 239.] BALTIMORE, FEBRUARY, 1911. [Price, 25 Cents

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LECTURES ON THE HERTER FOUNDATION.*

By Professor Hans Chiari, M. D.,

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I. The Relation of the Amnion to the Origin of Human Malformations.

If the pathologist would discuss the relation of the amnion to the origin of human malformations, he must base his views upon evidence gathered from pathological anatomy. He must study carefully the cases in point that he may recognize the variety of influences which the amnion can exert. He must seek industriously to ascertain causes for these pathological influences. Finally, from his own experience and that of others recorded in the literature, he must gain a correct view concerning the frequency of deformities which depend upon the amnion and must contrast them with deformities which arise in a different way.

Certainly, no one denies that human malformations may originate in other ways and quite independently of the amnion. Thus, we know a child may inherit a deformity from either its paternal or its maternal ancestors: polydactylism illustrates that fact. The cause for such malformations exists in the germ cells—either ovum or spermatozoon. Again, some disease of the embryo may occasion malformation which is exemplified by pathological collections of fluid within the body cavities (Höhlenhydrops). Occasionally, the umbilical cord or the second ovum where there is a double pregnancy, will exert a teratological influence. We also know that an injury directed against the abdomen of a pregnant woman may bring about fœtal malformation. To demonstrate the efficiency of this last influence I will relate the history of a case which I saw in the year 1881, while in Vienna, and which was published by Anton. The infant was a boy who died when fourteen days old. The autopsy revealed an imperfect corpus callosum; persistence of the embryonic median furrows of the cerebrum with microgyria; hair lip and cleft palate; and a fracture of the right femur which had healed in utero. The period at which the defect in the corpus callosum must have originated was the beginning of the fourth month of pregnancy. At this time, moreover, according to the mother's history, she had sustained an injury, falling on the ice in such a way as to receive the brunt of the blow upon her abdomen. This had caused the fracture of the femur, and favors Anton's view, that the same injury caused not only breaking of the bone, but also malformation of the brain. The pathologist who studies human malformations of amniotic origin, must do so in the light of both comparative pathology and experimental teratology. But he will be very careful in adopting their teaching, since the origin and significance of the amnion are not the same in man and in the lower animals.

*Delivered in the Medical Department of the Johns Hopkins University, October 5, 6, 7, 1910.

I have had opportunity to study an unusually large number of human malformations. The cases, I shall bring to your attention are selected from this material, which was secured at the Universities of Prague and Strassburg.

By far the most common human deformities of amniotic origin are those caused by "Simonart's bands." These string-like portions of amnion usually pass from that membrane to the embryo. A great deal has been written about the malformations they cause. Such deformities represent widely different types, including hair lip, cleft palate, micrognathia; fissure of the face, of the brain, of the spinal cord; fissure of the thorax and of the abdomen; constriction of the penis, and malformation of the extremities.* These bands run not only from the amnion to the fœtus, but may pass from one part of the surface of the fœtus to another. It has been satisfactorily demonstrated that subsequent development of the embryo or fœtus may cause the amniotic bands to disappear after they have produced deformity; hence their previous existence may be attested at the time of birth only by the damage they have worked.

I am indebted to Professor Kretz for permission to use the following cases from the Pathological Museum at Prague:

Fig. 1 shows both hands of a nine-month fœtus with perodactylia caused by Simonart's bands; in other respects it was normal. On the left hand the fourth and fifth fingers, on the right hand the third, fourth and fifth fingers are imperfectly formed; the fingers mentioned on the right hand are not separated from one another. Remains of amniotic threads are seen on both hands, adherent to the affected fingers. We were unable to examine the placenta.

I have found the same kind of deformity in a woman seventy years old, and in her case the deformity was certainly caused by amniotic threads (Fig. 2). The right hand presents atrophy of the fourth and fifth fingers; the left hand, atrophy of the fourth and fifth fingers, together with a defective nail on the fifth finger, a deep dorsal furrow on the fourth finger, a rather deep furrow on the back of the first phalanx of the third finger, and a furrow on the back of the second phalanx of the index finger. These deformities were all present at birth.

In proof of the fact that amniotic bands may cause constriction about more proximal parts of the extremity, I offer, as an example, the case pictured in Fig. 3, which is that of an infant who died when three hours old. It presented fissures of the brain and of the face. The left hand showed indentations upon the third, fourth and fifth fingers; absence of the nail phalanx of the fifth finger, at the ulnar edge of which there still hung a remnant of an amniotic thread. On the right side, and about 1 cm. below the elbow, a deep circular fissure was found; the constriction reached the neighborhood of the bones of the forearm. At this point the skin was rent and the bones were broken as the result of injury at the time of birth. Distalwards from this circular fissure were found the remains of an extremity. It measured 5 by 3½ cm., had an oval shape, and was covered with skin. This structure corresponded to the upper arm and the hand. It was markedly œdematous, and at its peripheral end bore four digits; three of them, namely, the fourth and fifth fingers and the thumb, were well formed, except that the nail on the last was lacking. All were of normal size. The second finger was represented by a short, pyramidal projection of skin without any nail, and had been drawn upwards and outwards to the side of the thumb. Amniotic threads joined the above mentioned constriction in the forearm to the rudimentary finger, and held the latter as would a sling. The third finger was entirely lacking.

It would have been only a step from this deep circular fissure formation to complete intrauterine amputation of the right forearm. In this instance also, we had no opportunity to examine the placenta.

Much less frequently, amniotic bands are found attached to other parts of the body. Fig. 4 shows a shrivelled, sixth-month fœtus papyraceous, which had died sometime before birth. Amniotic adhesions appear on the head. A band 4 cm. long and 2 mm. thick extended from the skull of the fœtus to a point on the placenta near the insertion of the umbilical cord, and was twisted in a spiral about the latter structure. A club-shaped appendage was found in the region of the large fontanelle, immediately in front of the amniotic band with which it was connected. The appendage was 1 cm. long, and 5 mm. thick at its free end. It was covered with skin, contained a cavity which could be traced through its pedicle into the cavum cerebri, and clearly represented an encephalocele. The periphery of the placenta had involuted and completely atrophied; but for that, the amnion presented its normal characteristics.

In this case, then, we have seen an amniotic band attached to the head of an embryo and an encephalocele produced through the traction thus occasioned. Moreover, fœtal death was very likely the result of its action, since constriction of the umbilical cord by the band would seem to account for that event.

Figure 5 represents a case of schizosoma caused by amniotic bands. The fœtus was about 38 cm. long. A short, ribbon-like band of amnion passed from the region of the navel to the back of the fœtus and had drawn along a tongue-shaped projection of skin with a small, round cutaneous appendage upon it. In consequence of this band, several deformities had arisen, namely, a deep transverse furrow across the back; imperfect closure of the pleuro-peritoneal cavity on the right side; protrusion of the viscera; and defective formation of the right upper extremity. There was no umbilical cord. The umbilical arteries and the umbilical vein issued from the lower edge of the abdominal fissure. These vessels were closely approximated, and ran to the placenta surrounded by a very small quantity of Whartonian jelly, which in turn was encased in amnion. The other malformations noted in this specimen were a median fissure of the thumb; talipes varus with heptadactylia on the left side and talipes valgus with hexadactylia on the right. A few amniotic threads hung upon the right

* See Von Winckel.

foot. The amnion which covered the placenta exhibited a few similar threads, scattered here and there over its inner surface.

Figure 6 illustrates the group of cases in which amniotic bands pass from one part of the fœtal body to another. This fœtus was 40 cm. long and of the male sex. An encephalocele, with a horizontal circumference of 22 cm. appeared upon the skull. The skin covering it was very thin; the hernial opening was 2.5 cm. wide; the cavity within the skull was quite small. A chilo-gnatho-palatoschisis appeared upon the right side of the face. From the chiloschisis a fissure ran upwards through the right half of the nose to the anterior edge of the hernial opening. A thread-like cord 10 cm. in length was attached at the latter point, and ran toward the right axilla, where it divided into three parts, one of which was attached to the anterior, and one to the posterior axillary fold; the third was attached to the back near the lower angle of the right scapula. Furthermore, the right hand was deformed; the fingers from the second to the fifth were small, malformed, without nails and constricted by manifold, delicate amniotic threads. The last joint of the fourth finger on this hand had turned black. The right foot showed a valgus, and the left a varus position. The placenta was not obtained.

Pathological anatomy also teaches that the mediation of Simonart's bands is not the only way in which the amnion may be causally related to fœtal malformations; abnormality in the size of the amniotic cavity, and particularly a diminution thereof reacts harmfully upon the development of the embryo. Cases of this sort have been recorded by Marchand and by Schwalbe. In the case reported by Marchand, the embryo had attained six to seven weeks' development. Adhesions were present, but there was also a particularly defective amnion. It was through the latter anomaly that the embryo had suffered extensive deformation. A continuous layer of amniotic epithelium was seen upon the inner surface of the chorion. Marchand attributed this either to a late proliferation of the epithelium from the point where the amnion had ruptured, or to cleavage between the amnion and the chorion. Schwalbe's case pertained to a seven-month fœtus, in which abnormal contracture of the amnion had caused unusual bending of the body, extensive adhesions of different parts of the body, and a series of malformations.

In this connection, I am able to add another case, illustrated in Figs. 7a and 7b. This specimen consisted of a 34 cm. long, female fœtus, which presented fissure of the face, a spur together with two cylindrical cutaneous appendages upon the hydrocephalic skull and perodactylia of both hands as well as of the right foot; from the digits of the last remains of amniotic adhesions hung. Moreover, in this specimen we found a fissure involving the abdomen and the lower part of the thorax. Most of the abdominal organs and the left lung projected through the opening. The trunk of the fœtus was conspicuously narrow, and gave the impression of having been squeezed in a corset. A thick, net-like covering of amnion, presenting many apertures, was fitted closely about the body, yet was not adherent save at the edges of the fissure. The umbilical cord entered at the right edge of the abdominal fissure. This structure was 13 cm. long, and was inserted upon the margin of the placenta, which had a diameter of 17 cm. The amniotic covering of the placenta, and indeed of the entire inner surface of the chorion was lacking. Microscopical study verified that fact, showing only a few delicate thread-like remnants of the membrane. The short umbilical cord was entirely covered by amnion.

From the findings in this specimen, the conclusion is unavoidable that the schizosoma had been produced by diminution in the size of the amnion. The body of the embryo had been surrounded by a closely applied corset which interfered with the closure of the pleuro-peritoneal cavity. The membrane had become detached in the neighborhood of the head and the extremities; these regions were separated from the rest of the amnion. Malformation of the head and extremities had been caused by Simonart's bands, remnants of which were attached to the inner surface of the chorion.

I found a specimen in the Prague Museum which probably belongs to this group of cases. The fœtus was a male, and had reached the fourth month of development. One could not actually demonstrate that the amniotic cavity had been contracted, but could prove that the cause of the deformity was abnormal pressure against the amnion in the locality of the affected parts (Fig. 8). The left upper extremity of the fœtus was rudimentarily developed, and supplied with only one digit. The limb was attached in a deep fossa upon the left wall of the thorax where the cartilages of the third, fourth and fifth ribs were absent, and the pectoralis major muscle, as well as the neighboring intercostal muscles, were very thin. In this instance, it seems fair to suppose that the amnion which enclosed the left upper extremity had been too small, and that perobrachia was produced in that way. Ritter, Ganghofner and Eppinger have described cases of the same character.

Viewed from the standpoint of pathological anatomy, it is not impossible that malformations and even death of the embryo may result from an abnormally spacious amnion, or to a premature collection of amniotic fluid. Yet, actual observations, so far as I know, have not been made to substantiate this opinion; nor have I observed cases which would seem to belong in that category.

What do we know concerning the cause and mode of formation of either Simonart's bands or of an abnormally small amniotic cavity?

Originally, Simonart's bands were thought due to an organization of lymph (Montgomery), which was referred to some inflammatory process (Simpson, Gratzer, Simonart). Many authors, including Virchow, accepted the inflammatory hypothesis. In 1854, G. Braun expressed the view for the first time that Simonart's bands were in reality abnormally disposed portions of the amnion itself. He inferred that they originated in consequence of pathological folding of the amnion, and that this phenomenon, in turn, depended upon a scant quantity of amniotic fluid.

C. Braun explained the presence of these bands through the formation of plastic adhesions. Kustner elaborated the theory,

and suggested that intimate contact was established between the amnion and the embryo during the early days of development. He considered that narrowing of the amnion, a deficiency of amniotic fluid and the formation of folds in the membrane might bring about adhesions between it and the young embryo. These adhesions gradually became firmer and occasioned the malformation. Possibly, other outgrowths might later develop from the amnion, but in that event the epithelium covering the surface of the fœtus would have to die before adhesions could occur. Finally, Kustner believed that a deficiency of amniotic fluid made it possible to have secondary adhesions arise in connection with fissure formation when the epithelium of the medullary folds or of the intestinal wall was elevated. Later Kustner described a case of congenital amputation of hands and feet by amniotic threads, which resulted from a rent in the amnion due to the mother lifting a heavy load during the second month of pregnancy. The amnion had entwined itself about the umbilical cord, and entangled the extremities of the embryo, thereby affecting their amputation. Olshausen discussed tearing of the amnion as a source of amniotic threads, and concluded that the amnion may rupture spontaneously if its cavity be too small. Stoeckel has quite recently collected a series of these cases in which extra-amniotic pregnancy was associated with amniotic bands.

My own view is that adhesions between the epithelium of the amnion and the ectoderm of the fœtus are of primary importance in the formation of Simonart's bands. Although we are not as yet sufficiently familiar with the development of the amnion (Graf v. Spee holds the interesting view that the amniotic cavity in the human arises as a closed cavity within the blastodermic vesicle and develops from the ectoblast) still we know that it is formed very rapidly, since the youngest human ova which have been described already possess a closed amnion. Embryos of 10 mm. are closely surrounded by amnion. Embryos of 11-15 mm. are separated from the amnion by a distance of only 1-3 mm., and it is only when a length of more than 20 mm. has been attained that the quantity of amniotic fluid becomes commensurate with the size of the embryo, thus insuring an appropriate separation between it and the amnion (Marchand, Kollman).

With these facts in mind, it is not difficult to understand how the human embryo may become adherent to the amnion during the early weeks of pregnancy. The ectodermal cells on the inner surface of the amnion, as well as those covering the surface of the embryo, are young and active, and can fuse together easily if they are not sufficiently separated from one another. The union will gradually grow firmer. Later, when the amniotic fluid begins to develop, the adhesions are drawn out and become Simonart's bands, stretching from amnion to fœtus, or from one part of the fœtus to another. Inasmuch as these adhesions occur during the earliest stages of development they cause the most extreme types of deformity, one can imagine. This conception, which includes a primary adhesion and a later fusion of the two epithelial surfaces belonging respectively to the amnion and to the embryo, agrees perfectly with the histological findings in Simonart's bands. Microscopically, these bands consist of a wavy mass of amniotic connective tissue, exhibiting very few nuclei and no blood vessels. They are covered by amniotic epithelium which shades into the fœtal epidermis. As they approach the embryo, the covering of these bands often presents the typical structure of skin, from which we may infer that they ultimately draw out the skin, making it a part of the band.

In addition to the mode of formation just described, others occur, though much less frequently: thus, Simonart's bands may arise in consequence of rents in the amnion. This appears to me to support the interpretation of the previously mentioned case with corset-formation, which was attributed to contracture of the amnion combined with laceration of the membrane and the production of Simonart's bands.

Valuable contributions from the field of experimental teratology shed light upon the causation of narrowing of the amnion, and the mechanism by which it is produced. In this connection, reference should be made to the unusually interesting experiments of Dareste. By raising and lowering the temperature of an incubator, and by warming different parts of a hen's egg unequally, this investigator frequently succeeded in producing disturbances in the development of the amnion. Moreover, embryonic malformations, apparently of amniotic origin, were noted: cyclopia was occasioned by narrowing of the head-fold, and symelia by narrowing of the tail-fold. These experimental observations support the conception that narrowing of the amnion has a very direct bearing upon the origin of certain human malformations. To be sure, these results are not strictly analogous with similar human deformities, since the formation of the amnion in the chick and in man are essentially different. In the former, the amnion is formed by the union of the folds which arise at the anterior and posterior ends of the body as well as at either side. These folds meet and fuse over the back of the embryo. With this mechanism, it becomes very easy to have serious interference with development of the embryo, since either the head, the tail, or one of the side folds may not rise sufficiently, and thus fail to reach its proper destination. As a result of such aminotic defects, malformation of the embryo occurs.

In the development of the human ovum, according to His, the amnion is formed exclusively by the head-fold; this extends over the embryo toward the tail, and ultimately shuts in the amniotic cavity. None the less, here again it becomes entirely possible for the amnion to attain incomplete development through one harmful influence or another, and a pathological narrowing of the cavity may result. We can also imagine the amnion becoming abnormally firm and rigid in consequence of some disease; so that it is incapable of the distension requisite for the subsequent growth of the embryo. Of course that is only an hypothesis at present. We meet occasionally with cases which demonstrate that the amnion may undergo pathological thickening, and attribute it to an inflammatory change. In this connection, the interesting case of v. Franqué should be cited. There a number of wart-like nodules were noted on the fœtal side of the amnion, and near them superficial plaques were found. These had arisen through the proliferation of amniotic stroma about certain

products of the fœtal skin (vernix caseosa, desquamated epidermis, small clumps of lanugo hair), which had been deposited upon the amnion. Further, the fact should be mentioned that so-called "wind eggs"—a name given cases in which the embryo has perished—often present circumscribed or diffuse thickening of the amnion. Microscopically, the condition proves to be a proliferation of amniotic stroma and epithelium.

What proportion of human malformations can be fairly ascribed to an amniotic origin may be difficult to decide, but no doubt can exist that the number is large. Among 178 cases of malformation studied by v. Winckel, 29 cases (16.3 per cent) were referable to Simonart's bands. My own experience leads me to a similar conclusion. Beside this group, we have the other in which deformity is due to narrowing of the amniotic cavity; and the latter, according to my view, is at least as large as the former. Combining the two, I would attribute one-third of all human malformations to some influence exerted by the amnion. Such an estimate, moreover, does not take into account the deformities met with in cases of hydramnios.

Oftentimes, it becomes impossible to demonstrate the amniotic origin of malformations. On the one hand, it frequently happens that the membranes are studied too superficially, and on the other, Simonart's bands, as well as evidence of the narrowing of the amniotic cavity, may disappear in the latter months of pregnancy. For these reasons, it is desirable to collect a larger amount of material and study it more thoroughly than has been done.

In conclusion, I should like to speak of congenital defects, which occur in the skin, especially about the head, and have been referred to the traction of amniotic bands. Keller has recently reviewed cases of this sort and included a new example, which was observed in the Pathological Institute at Strassburg. The defect was located upon the skull, and differed, in one respect, from similar cases thus far reported. Microscopical examination in this instance showed that hair follicles were present at the site of the defect. That finding does not agree with the hypothesis that very early amniotic adhesions existed. Therefore, Keller, concluded, if the tearing loose of amniotic adhesions was to account for the deformity in his case, the adhesions must have developed after the fourth month and not during the early days of embryonic development. Moreover, he held that some inflammatory process was fundamentally responsible and his opinion had support in that the mother had suffered from endometritis. The placenta was firmly attached to the uterus and its separation was accomplished with difficulty. Such a case certainly suggests that inflammatory changes in the amnion be given careful consideration in future study of the development of the ovum.

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II. NECROSIS OF THE PANCREAS.

Necrosis of the pancreas may arise from many different causes. It may be due to a severe trauma, directly affecting the organ, as for example in gunshot wounds; or it may be caused by corrosive poisons, such as sulphuric acid, acting through the stomach wall. Again it may develop from the perforation of a petic ulcer of the stomach, or result from an inflammation of the pancreas, in so far as extensive necrosis and putrefaction may follow suppuration of the organ. It is my belief, however, that much the most common cause of necrosis of the pancreas is autodigestion due to the tryptic ferment of the pancreas.

The corrosive action of the pancreatic secretion has been known for a long time, and the tryptic autodigestion of the pancreas has been pointed out likewise. In 1879, Klebs attributed pancreatic hemorrhages to the corrosive action of pancreatic juice. Gussenbauer in 1883 described a cyst of the pancreas which had been formed by the destruction of a melanosarcoma by the pancreatic secretion. Arnozan and Vaillard referred in 1884 to the post mortem autodigestion of the pancreas in animals. Selzer (1886) and Wölfler (1888) thought that the erosion of the walls of pancreatic cysts by the pancreatic secretion might lead to hemorrhages. Pelliet (1889) and von Hansemann (1894) noted the frequency of post mortem autodigestion of the pancreas. Tilger (1894) attributed the origin of pancreatic cysts to autodigestion in pancreatitis. Nimier (1894) emphasized the importance of the pancreatic secretion as a source of pancreatic cysts, following injury to the duct of Wirsung. In 1895 I described the occurrence of intra-vitam, intra-agonal and post mortem auto-

digestion of the pancreas, and drew a comparison between this and peptic autodigestion of the stomach wall. Blume (1897) and Pförringer (1899) supported my views, and Blume, as well as Beneke, sought the cause for intra-vitam autodigestion in temporary ischæmia, and Pförringer in fat necrosis. I described in 1900 a case of focal intra-vitam autodigestion in all probability caused by arteritis obliterans. Gaylord (1901) reported autodigestion of the pancreas following gunshot wounds. In 1901 and 1902 Lazarus attributed anew the origin of pancreas cysts to autodigestion.

Numerous cases of intra-vitam tryptic autodigestion of the pancreas were, in my opinion, incorrectly interpreted, being described as hemorrhage of the pancreas, as pancreatitis hemorrhagica and gangrenosa (Fitz) and as necrosis of the pancreas from other causes. Here are to be found many pertinent observations, which resulted from investigation of cases of fat tissue necrosis by Balser (1882) which have since been proved to be due to a lipolytic ferment of the pancreas. The necrosis of the pancreatic tissue so frequently found has been considered, by most of the writers, as secondary in relation to the necrosis of the fat tissue, and even those authors who regarded the pancreas necrosis as primary, and the fat tissue necrosis as secondary, did not ordinarily think of a tryptic autodigestion as the cause of the pancreas necrosis, but rather assumed a primary pancreatitis. Only a few writers (Dettner, 1895; Hildebrand, 1898; Konheimer, 1898; Kaufmann, 1891) took the position that the spontaneous fat tissue necrosis was due to an intravital tryptic autodigestion, rather than to a traumatic rupture of the ducts of the pancreas. In 1902, I expressed myself firmly of the belief that both these conditions regularly belonged together, in the sense that the primary factor was the tryptic autodigestion of the pancreatic tissue, which was followed by the necrosis of the fat tissue, due to a pouring out of the fat ferment and its diffusion. I was also able to show that in post mortem and especially in intra-agonal autodigestion of the pancreas, it was not seldom the case that the fat tissue in the pancreas showed the beginning of the changes as they occur in necrosis of the fat tissue, and I explained further that the pancreas hemorrhages and pancreatitis acuta hemorrhagica and gangrenosa were nothing more than changes in the pancreas due to autodigestion. Necrosis is primary and possible inflammation is secondary. Then Weil (1904), Reitter (1905) and Truhart (1906) expressed similar opinions. In 1906 I maintained the view that the intra-vitam tryptic autodigestion of the pancreas might, *on the one hand*, be a most important process, leading to extensive hemorrhages in the pancreas and its neighborhood, to hemorrhagic cyst formation of the pancreas, to focal or diffuse necrosis of the pancreas and to suppurative pancreatitis and sequestration of the pancreas if pathogenic bacteria invaded it; but that *on the other hand* from circumscribed slight tryptic autodigestion of the pancreatic tissue, harmless, small areas of pancreas necrosis and fat tissue necrosis (secondary autopsy findings) might develop, which areas of pancreas necrosis might disappear entirely by absorption. But in view of this, tryptic autodigestion of the pancreas deserved the title of a *morbus sui generis*. Williams and Busch (1907), Truhart (1908) and most recently, Opie and Meakins (1910), have expressed similar views, so that the doctrine of the tryptic autodigestion necrosis of the pancreas seems to be well founded.

There is not the least doubt that the *causes for the occurrence of a "spontaneous" intra-vitam tryptic autodigestion* of the pancreas may be manifold. Anything which may lead to injury of the tissues of the pancreas or to activation of the tryptic zymogen, may induce tryptic autodigestion of the pancreas. I would call especial attention to the following important etiological factors: slight trauma of the pancreas, not causing rupture of a duct; disturbances of the pancreatic circulation, which may be looked upon (Benecke and Blume) as spastic anæmias, which may be caused by the frequently noted arteritis obliterans; stagnation of the pancreatic secretion; entrance of bile into the duct of Wirsung in cases of cholelithiasis, as was first shown by Halsted and Opie in 1901; and finally entrance of the secretions of the stomach, or the contents of the duodenum into the duct of Wirsung (Hess, 1903 and 1905; Beitzke, 1905). In individual cases one must always recall, and consider, as Eppinger showed in 1905, that the kinases which are able to activate the pancreatic secretion, may be of many different kinds. I will not deny that tryptic autodigestion of the pancreas may arise from a primary inflammation of the organ, but I think it happens rarely.

The consequences of tryptic autodigestion of the pancreas may vary widely. As I have mentioned, a grave disease may result. Death may result from severe hemorrhage—the so-called apoplexy of the pancreas. Within a few days death may follow after symptoms of ileus and peritonitis, when one usually finds extensive necrosis of the fat tissue in association with the reactive inflammatory necrosis of the pancreas. The cause of death in such cases is to be attributed either to a resorption of a pancreas toxin (Dobenauer, 1906) a product of necrosis, or to poisoning by trypsin (Guleke, 1906). Starting from the pancreas a diffuse abdominal tryptic destruction of the cellular tissue may arise, as I, myself, had opportunity to report in a case (1909). Suppuration and gangrene of the pancreas, with eventual sequestration of the organ, may develop. Cysts of the pancreas may form in consequence of the tryptic autodigestion. On the other hand, however, the tryptic autodigestion of the pancreas may be an insignificant process, which induces small circumscribed necrosis of the tissue of the pancreas, and small circumscribed areas of fat tissue necrosis. In the course of time all these foci may disappear, through absorption of the necrotic tissue, or at least of the foci of (necrotic) pancreatic tissue in which case we may conclude from the presence of old fat necrosis in the pancreas and its neighborhood that there has been an earlier autodigestion of the gland.

From all this it is easily seen what an important rôle the tryptic autodigestion of the pancreas plays. It is surely one of the most important factors in the pathology of this organ. The practicing physician must be cognizant of this autodigestion, since experience has shown that in the severe cases life oftentimes may be saved by surgical measures.

III. SPONDYLOLISTHESIS.

Following the enunciation by Kilian in 1853 that spondylolisthesis is a pelvic deformity resulting from a gradual gliding forward, or an olisthesis, of the inferior surface of the last lumbar vertebra over the anterior border of the first sacral vertebra, a large special literature has developed upon the subject.

The spondylolisthetic pelvis is of the greatest practical interest to the obstetrician on account of the part which it may play in the production of dystocia. On the other hand, the olisthesis demands attention from the pathological anatomist as an example of a peculiar acquired displacement of the vertebræ, whose mode of origin is not easy to understand.

Practically every case coming within this category has been described and published, so that Neugebauer, who devoted particular attention to its study was able to collect

11	clinical	and	12	anatomical	cases	in	1882
37	"	"	24	"	"	"	1885
47	"	"	53	"	"	"	1892
and 55	"	"	60	"	"	"	1893,

making a total of 115 cases. This, however, included 10 cases of olisthesis of the fourth lumbar vertebra, and one instance involving the first sacral vertebra. Furthermore, in 1899, Williams described a new specimen, and collected 123 cases from the literature, 62 of which were clinical and 61 anatomical. He also stated that Dr. Authur Dean Bevan had informed him that he had observed several cases in the dissecting room in Chicago.

According to Olshausen and Veit about 130 cases had been reported up to 1902, 70 of which were described anatomically and the remainder reported clinically.

Following the publication of Williams, and including his specimen, I have been able to collect 10 clinical and 7 anatomical cases, so that the entire number thus far recorded amounts to 146. At the same time, it must be admitted, particularly among the cases which were described clinically, that in not a few the diagnosis was at least doubtful. Moreover, in several specimens, which were described anatomically, only the pelvic portion of the vertebral column or even isolated vertebræ were available for study, so that Breus and Kolisko in 1900, in their monumental work on Pathological Forms of the Pelvis, after excluding the imperfect preparations, as well as those in which the olisthesis was only beginning, stated that only 18 examples of well-developed spondylolisthesis were on record, accompanied by a satisfactory anatomical description of the pelvis, and to these they added two additional specimens which they had discovered in Vienna.

Especially from a clinical point of view, the means of diagnosticating spondylolisthesis have been fully developed; and a series of characteristic symptoms described, such as the transverse furrow in the back above the lumbo-sacral juncture, the lumbar lordosis, the diminished pelvic inclination, and the peculiar "rope dancer gait" of the individuals affected by it. At the same time, the importance of lumbo-sacral spondylolisthesis has been accentuated on account of the important part it may play in reducing the size of the pelvis and thus interferring with the act of labor.

Anatomically the various changes in the shape of the bones and ligamentous structures have been described, and the effect of the olisthesis upon the form of the pelvis has been studied, more particularly by Breus and Kolisko. According to these authors, a fully developed spondylolisthetic pelvis is a "pelvis obtecta," which is composed of plump, massive bones. With the exception of the conjugata vera, the pelvic measurements are usually large. The former, however, as a result of the olisthesis, is markedly shortened, in that the antero-posterior diameter of the pelvic inlet, which takes the place of the vera, will impinge posteriorly upon the ventral surface of one of the prolapsed lumbar vertebræ, instead of upon the promontory of the sacrum. The pelvic inclination is also diminished, the upper end of the sacrum appears to be inclined posteriorly, and the region of the symphysis pubis elevated. Frequently, the vertical concavity of the sacrum is markedly increased and the entire bone is retroposed, and the articulation between the first and second sacral vertebræ gapes toward the sacral canal. The ilio-sacral joints are markedly developed, their ligaments strong and the acetabula deep. All of these changes are to be attributed to the forward displacement of the center of gravity of the body, and to the change in the manner in which the body weight is transmitted to the sacrum, and tend to become more and more pronounced until the olisthesis is checked by synostotic processes.

The most interesting question in connection with this subject is the mode of production of the olisthesis, and numerous theories have been advanced in the attempt to solve it. Kilian in his original communication considered that the most important etiological factor consisted in an inflammatory softening of the last intervertebral cartilage, as well as of the neighboring ligaments and vertebræ. At the same time, he admitted the possibility of spondylolisthesis developing as a result of retrogressive changes in the first sacral vertebra. Ritgen in 1854 expressed the belief that the anomaly might result from an œdematous condition of the spinal membranes, which had not led to the production of spina bifida (hydrorrhachis incolumnis).

Robert in 1855 was the first to resort to experiment in the hope of elucidating the etiology of lumbo-sacral spondylolisthesis, and showed that it never followed mere section of all the ligamentous structures about the lumbo-sacral juncture, but readily occurred after the interarticular portions of the arch of the last lumbar vertebra had been sawed through, and the last intervertebral cartilage severed. From these observations, he concluded that the development of lumbo-sacral spondylolisthesis is usually prevented by the articular processes, and can only occur after their destruction. Or, it might follow luxation of the inferior articular processes of the last lumbar vertebra, or finally be due to a general softening of the ring of the last lumbar vertebra which permitted its articular processes to become so far removed from its body that the latter could glide forward and downward, while its inferior

articular processes still remained in contact with the superior articular processes of the first sacral vertebra.

The hydrorrhachis theory was generalized by Lambl in 1856 and in 1858. He held that such a condition could give rise to a pseudoarthrosis of the interarticular portion of the last lumbar vertebra, which would lead to the formation of a spondylolysis interarticularis. At the same time, he admitted the possibility of spondylolisthesis lumbo-sacralis developing as the result of luxation of the lumbo-sacral articular processes, or from a fracture of the arch of the last lumbar vertebra, as well as from the presence of a rudimentary accessory vertebra (Schaltwirbel).

Breslau, in 1861, in describing a new case, designated as caries sicca an inflammatory softening of the bony tissue of the last lumbar and first sacral vertebræ, as well as of the last intervertebral cartilage; and stated that the lengthening of the vertebral arch in spondylolisthetic processes could therefore be secondary in nature. In 1863 Hugenberger attributed a case of traumatic lumbo-sacral spondylolisthesis to forcible luxation or fracture of the articular processes; while Blake in 1867 considered that the development of the olisthesis might be due to excessive stretching of the ligaments at the lumbo-sacral juncture resulting from lipomatosis universalis. Blasius and Ender in 1868 and 1869, respectively, contended that their cases should be attributed to lumbar caries.

The idea that spondylolisthesis lumbo-sacralis might be the result of abnormal stretching of the interarticular portions of the last lumbar vertebra, dependent upon imperfect ossification, originated with Königstein in 1871, and was based upon experiments similar to those of Robert, to which reference had already been made. At the same time, he admitted the possibility of Lambl's theory, and of the part played by an accessory vertebra. Von Weber-Ebenhof in 1874 described two cases, which he considered followed luxation of the lumbo-sacral articular processes, and contended that this was the general rule. Caries of the lumbo-sacral articular processes was considered by Didier in 1874, and by Herrgott in 1877, as a satisfactory explanation for the development of spondylolisthesis, and Van der Bosch expressed similar views in 1879.

In 1878 I contended that the cause of the olisthesis in the "kleines Wiener Becken" was to be found in a congenital anomaly of the sacrum, while in the "grosses Wiener Becken" the lesion in continuity in the interarticular portions of the last lumbar vertebra was due to fracture.

In 1882 Strasser believed that his case of lumbo-sacral olisthesis was the result of arthritis deformans of the lumbo-sacral joints, with the consecutive elongation of the arch of the last lumbar vertebra.

In the same year, appeared the highly important monograph of Neugebauer, in which he demonstrated that in lumbo-sacral spondylolisthesis only the anterior half of the last lumbar vertebra showed signs of displacement, and that the "dolicho-platy-kyrto-spondylus" which characterized it was the result of imperfect ossification of the interarticular portions of the last lumbar vertebra, or was due to their fracture. He accordingly defined spondylolisthesis as a mechanical deformity (Belastungs-difformität) of the last lumbar vertebra, developing gradually in extra-uterine life in the presence of certain predisposing conditions, as the result of the action of the body weight. Moreover, in 1852, Neugebauer stated on the basis of Strasser's case that an originally normal last lumbar vertebra might develop this deformity secondarily, as the result of an olisthesis due to fracture of the superior articular processes of the first sacral vertebra, should the former be arrested by the posterior margin of the first sacral vertebra and acquire an abnormal chronic inflammatory plasticity. Under such circumstances, a further olisthesis could develop only from the anterior half of the last lumbar vertebra.

Neugebauer reiterated similar views in 1883, 1884 and 1885, and laid particular stress upon the frequency of interarticular spondylolysis, which he gradually came to attribute more and more to a primary defect in development. In 1884 and 1885 Arbuthnot Lane dissented from Neugebauer's teaching concerning the great importance of congenital spondylolysis interarticularis, and contended that spondylolisthesis was merely a manifestation of a deformity of the last lumbar vertebra, due to excessive pressure resulting from the continued carrying of heavy burdens by the individual. This he held not only gave rise to the change in form of the vertebra, but also to the lesions in continuity in the interarticular portions.

Kruckenberg in 1885 pointed out that fracture of the lumbo-sacral articular processes might also occur secondarily in the course of an olisthesis; and H. von Meyer in 1877, after studying a case of spondylolisthesis of the first sacral vertebra, stated that the condition might occur as the result of imperfect development of the superior articular processes of the first sacral vertebra, even without the presence of spondylolysis interarticularis.

Neugebauer described a new spondylolisthetic pelvis in 1889, and demonstrated that it was certainly not attributable to hydrorrhachis, and clearly refuted the attacks of Lambl in that connection. In the following year he also adduced conclusive evidence of the possibility of a separation in continuity in the interarticular portion of the last lumbar vertebra occurring as the result of trauma, and gave a very accurate anatomical description of such a case.

In 1892 I attempted to bring together the various possible etiological factors concerned in the production of spondylolisthesis, and based my conclusions upon a careful study of the literature, the investigation of a comparatively large personal material, as well as upon experimental research. My investigations led me to define spondylolisthesis lumbo-sacralis as a gradual pushing forward of the last lumbar vertebra over the base of the sacrum, when the change might involve the vertebra *in toto* or only in its anterior half.

As a result of my studies, I held that any one of the following conditions might be considered *a priori* as etiological factors.

I. In olisthesis of the entire last lumbar vertebra.

1. Developmental anomalies of the lumbo-sacral articular processes.

2. Destruction by disease of the lumbo-sacral articular processes.
3. Fracture of the lumbo-sacral articular processes.
4. Luxation of the lumbo-sacral articular processes.

II. In olisthesis of the anterior half of the last lumbar vertebra.
1. Developmental anomalies of the arch of the last lumbar vertebra.
2. Softening or destruction by disease of the arch of the last lumbar vertebra.
3. Fracture of the arch of the last lumbar vertebra.
4. Changes in form resulting from carrying excessive burdens.

At that time I was able to adduce personal cases in support of the correctness of several of these hypotheses, more particularly for I, 1 and 3, and also for II, 1 and 3. As far as the others were concerned, I was obliged to state that they had been advanced merely as hypothetical or probable causes of the condition; as for example I, 4, but that their actual occurrence had not been demonstrated, nor did it appear likely that conclusive evidence in their support could be adduced in the future. Moreover, I was able to show in another specimen of pronounced spondylolisthesis lumbo-sacralis that it was impossible to determine which factors had been concerned in its production, when the synostosis at the displaced lumbo-sacral juncture was far advanced. Likewise it seemed important to me to point out that, even when the existence of one of the causal factors just mentioned could be demonstrated, it was necessary for excessive pressure on the part of the body weight to come into play in order for an olisthesis to develop from the predisposing etiological condition.

In 1892 Neugebauer reviewed the entire literature upon the subject, and considered all cases which had been either clinically or anatomically described up to that time. As a result, he stated that the important etiological factors were spondylolysis, which might be either congenital or the result of fracture, arthritis of the lumbo-sacral articular processes, and static deformation in the sense of Arbuthnot Lane. In the following year, he was able to enlarge his previous list by 8 additional clinical cases and 7 anatomical preparations, and he referred particularly to the highly interesting communication of Arbuthnot Lane in 1893, in which was described an attempt to cure spondylolisthesis, resulting from static deformation, by removing the spinous process and the arch of the fourth lumbar vertebra. This case is of particular interest as it represents the first attempt to treat spondylolisthesis by surgical measures.

During the following years a number of casuistical publications appeared, and I would mention especially the work of Williams, who, in 1899, described both anatomically and clinically, a well-marked case of spondylolisthesis occurring in a negress resulting from a spondylolysis interarticularis of traumatic origin, and which he stated was the sixth case which had been described in America up to that time. Moreover, I should like to call attention to the publication of Lawrence in 1900, who, after excluding the possibility of a lesion in continuity in the interarticular portions of the last lumbar vertebra being the etiological factor in his case, attributed it to imperfect development of the lumbo-sacral articular processes and of the arch of the last lumbar vertebra.

In 1900 Breus and Kolisko published their very thorough and systematic work upon spondylolisthesis, particularly from a pelikological point of view, to which I have already referred. Etiologically they considered that the typical factor in its production was spondylolysis interarticularis.

In 1905, I studied a new case of spondylolisthesis, in a 70-year-old woman, developing from congenital spondylolysis interarticularis of the last lumbar vertebra, and was able to demonstrate the presence of small accessory bones (Schalteknochen) in the space between the fragments of the left interarticular portion, just as I had repeatedly observed in simple congenital spondylolysis interarticularis without olisthesis. In 1901 Schwarte resuscitated the teachings of W. A. Freund, and contended that spondylolisthesis developing from congenital spondylolysis interarticularis was frequently due to the fact that the fœtal form of the pelvis had persisted, and consequently that the deformity was frequently of pelikogenous origin.

As the result of the many investigations concerning the etiology and pathogenesis of lumbo-sacral spondylolisthesis, I consider that it has been shown beyond peradventure that the etiological factor most frequently concerned is congenital spondylolysis articularis of the last lumbar vertebra. It has also been satisfactorily demonstrated that fracture of the interarticular portions of the last lumbar vertebra may sometime play a causal part, while occasionally, an unusually scanty development or fracture of the lumbo-sacral articular processes, as well as their involvement by arthritis, may be the predisposing cause.

When the lesion in continuity is limited to the interarticular portion of the last lumbar vertebra, only its anterior half glides forward, whereas the entire vertebra undergoes displacement in the other conditions. Moreover, when the vertebra becomes arrested in a certain location, the possibility must be considered that it may increase in length secondarily, in which event its body may later move further and further forward; just as we must admit that the flattening and lengthening of the interarticular portions of the last lumbar vertebra may be due to the action of undue pressure, as advocated by Arbuthnot Lane; but, personally, I consider it improbable that a marked olisthesis can develop in this manner. Spondylolisthesis occurring elsewhere than at the lumbo-sacral juncture is so very rare that it need not be considered at this time.

I believe that in the future our knowledge of spondylolisthesis will be advanced in great part by the study of pelves in which the anomaly is only slightly developed; as in them the etiological factors concerned can be much more clearly appreciated than in more advanced cases; since in the latter the presence of extensive synostoses may completely mask the underlying anatomical and etiological conditions. Such investigations will gradually place its etiology in a much clearer light, and will enable us to estimate more accurately

the frequency with which the various factors come into play. They will also have to take into consideration, to a much greater extent than previously, the part played by trauma in the development of spondylolysis, as well as by the mode of life of the individual. Finally, they will teach us whether spondylolisthesis really occurs so much more frequently in females than in males, as seems to be indicated by the statistics thus far available.

In order to solve these problems, it will be necessary to search directly and systematically for spondylolisthesis in all pathological anatomical material. At every autopsy, whether upon male or female subjects, the region of the lumbo-sacral juncture should be carefully examined, and, whenever it appears to be more prominent than usual, the pelvis should be removed and subjected to accurate preparation with a view of determining the existence of spondylolisthesis. Moreover, we should study all pelvic preparations, which are preserved in museums, with this point in mind.

In this manner I was enabled to discover three slight cases of spondylolisthesis lumbo-sacralis in my work in Prague prior to 1892, one of them being due to imperfect development of the lumbo-sacral articular processes, while in 1905 I described a fourth case. In about 4000 autopsies (25 per cent of which were upon children) performed under my supervision during the past four years in Strassburg, I discovered three additional examples of early spondylolisthesis. Etiologically they were quite ordinary, in so far as the causal factor was spondylolysis interarticularis; but on the other hand they were of especial interest in that they all occurred in males.

Up to 1893, out of a total of 115 cases, Neugebauer found only 7 examples of spondylolisthesis in males, an incidence of 6 per cent; Williams, in 1899, only 8 in 123 cases, 6.5 per cent; and, up to the time of this communication I could collect only 9 cases in 144, 6.2 per cent. If, however, I add to them the three cases to which I have just referred, we find 12 male cases in 147, and thus raise the incidence to 8.1 per cent. This being the case, it would appear permissible to predict that even in the male sex spondylolisthesis occurs much more frequently than is generally believed, and therefore the male pelvis must be examined with particular interest in this regard.

In this connection it is important to note that up to the present time all cases of spondylolisthesis occurring in males were only of slight degree, but whether this is due to the greater strength of the ligamentous structures and the more pronounced development of the musculature in that sex I am not yet prepared to state.

I shall now pass on to the description of my three new cases.

Case 1.—Museum specimen 6769 (Figs. 1 and 2). This specimen was obtained from a 70-year-old, very strongly built, peasant, 167 cm. in height, who died March 7, 1910, from embolism of the pulmonary artery. On removing the viscera from the abdominal cavity at autopsy, a marked protuberance was noted at the lumbo-sacral junction. Accordingly, after counting the normal number of cervical, dorsal and lumbar vertebræ, the pelvis was removed from the body and carefully prepared. On examination it was found that the spinous process of the 24th vertebra projected about 1 cm. further posteriorly than those of the other lumbar vertebræ, while its body was pushed forward to the same extent beyond the body of the 25th vertebra. A somewhat flattened exostosis 4 to 5 cm. broad extended from the anterior surface of the body of the 24th vertebra, principally to the left of the middle line, and was firmly united to a similarly formed exostosis of the superior border of the 25th vertebra by means of fibrous tissue. Moreover, a lip-like hyperostosis was present on the adjacent antero-lateral margins of the 24th and 25th vertebræ, while the intervertebral cartilage between them was markedly reduced in size.

On studying the relations of the 24th to the 25th vertebræ it was seen that the body of the former had advanced 1 cm. in front of the posterior border of the upper surface of the latter, and that both interarticular portions of the 24th vertebra presented a symmetrical spondylolysis interarticularis; the ends of the bone being smooth and about 1 cm. distant from one another, while the intervening space was filled by ligamentous structures, thus accounting for the increased length of the vertebra. The 25th vertebra presented signs of lumbo-sacral assimilation and was separated from the 26th by an intervertebral cartilage 8 mm. in thickness, while its alæ were only very slightly developed. On the right side, a suture separated the ala of the 25th from the normally developed ala of the 26th vertebra, while on the left side the two alæ were synostosed together; although a depression in the bone indicated their original union. Anteriorly and to the right, between the bodies of the 25th and 26th vertebræ, was a button-shaped exostosis, 4 cm. broad, 2 cm. long and 1.5 cm. high, which served to bind the two bones together. The spinous process of the 25th vertebra was split into a right and left half. The 26th vertebra was otherwise normal; as were also the 27th, 28th and 29th vertebræ. The 30th, however, appeared to be entirely assimilated to the sacrum and below it were three well-marked coccygeal vertebræ. Either side of the sacrum presented 5 sacral foramina, while the sacral hiatus gaped from the 28th vertebra downward. The sacro-iliac joints were not synostosed, and their anterior margins were lip-like. The inclination of the pelvis appeared somewhat less than normal and its bones were all strongly built and the acetabula were of moderate depth.

The pelvis presented the following measurements: Distance between iliac crests, 27.5; between anterior superior spines, 25.5; and between superior posterior spines, 8.5 cm. Superior strait: Conjugata vera, 10.5 cm.; transverse diameter, 12.5 cm.; right oblique, 12.5 cm.; left oblique, 12 cm.; right and left sacro-cotyloid diameters 8.2 and 7.8 cm., respectively. Plane of greatest pelvic dimensions: Conjugata, 13 cm. (extending to the junction between the 27th and 28th vertebræ); transverse diameter, 11 cm. Plane of least pelvic dimensions: Conjugata, 11.5 cm. (extending to the lower end of the 30th vertebra); distance between ischial spines, 9 cm.; distance between tubera ischii, 6.5 cm.

The sacral portion of the innominate bone was 8 cm. on each side (measured with a pelvimeter); the iliac portion was 6.4 cm. on the right and 5.5 cm. on the left side (tape measure), while the pubic portion measured 7.9 cm. on both sides. The sacrum measured 14 cm., from the upper margin of the 25th to the lower margin of the 30th vertebra, and 11.2 cm. in width.

From the study of this case, it appears that we have to do with a pelvis presenting a slight spondylolisthesis of the 24th vertebra resulting from an apparently congenital bilateral interarticular spondylolysis. This was combined with a lumbar assimilation of the 25th vertebra, and it is very interesting to note how this condition brought it about that the hypersynostotic processes, resulting from the olisthesis, involved not merely the junction between the 24th and 25th vertebræ, but also that between the 25th and 26th. The development of olisthesis in this case from the spondylolysis interarticularis may be explained by the hard labor which the man was obliged to perform, while further slipping

FIG. 1.

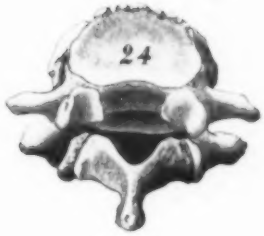

FIG. 2.

FIG. 3.

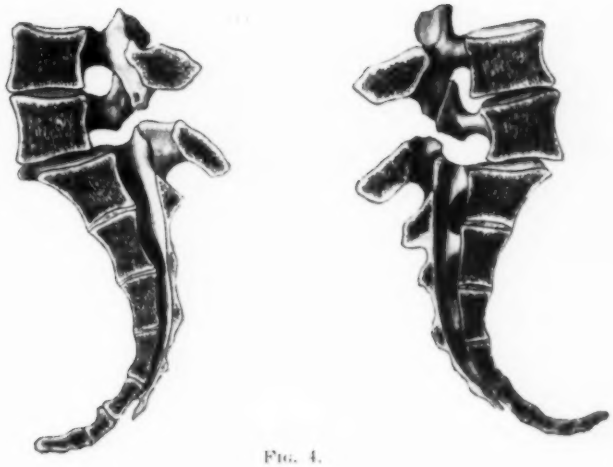

FIG. 4.

FIG. 5.

FIG. 6.

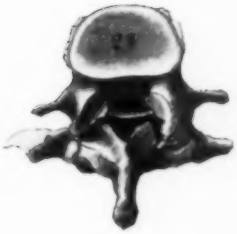

FIG. 7.

FIGS. 1 and 2.—Spondylolisthesis lumbo-sacralis e spondylolysi interart. cong. vert. lumb. V. Assimilatio lumbalis vert. sacr. I Strassburg. Mus. 6709. Male 70a.

FIGS. 3 and 4.—Spondylolisthesis lumbo-sacralis e spondylolysi interart. cong. vert. lumb. V. Strassburg. Mus. 6628. Male 55a.

FIGS. 5, 6, and 7.—Olisthesis vertebræ lumb. IV e spondylolysi interarticulari fracturosa. Strassburg. Mus. 6651. Male 55a.

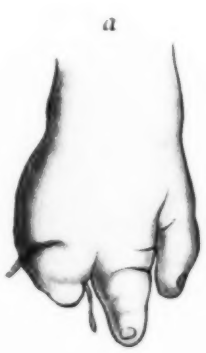

Fig. Ia.

Fig. Ib.

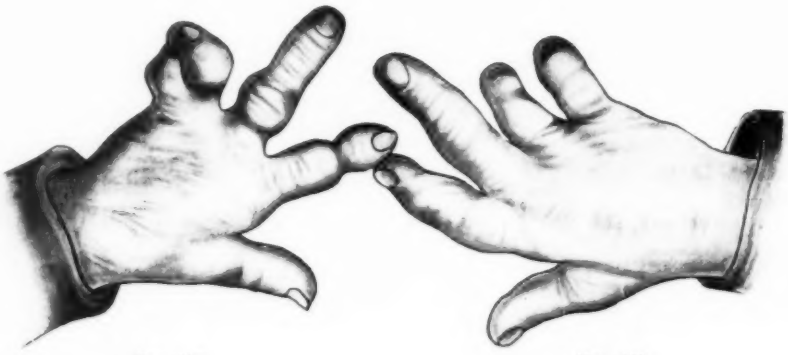

Fig. IIa. Fig. IIb.

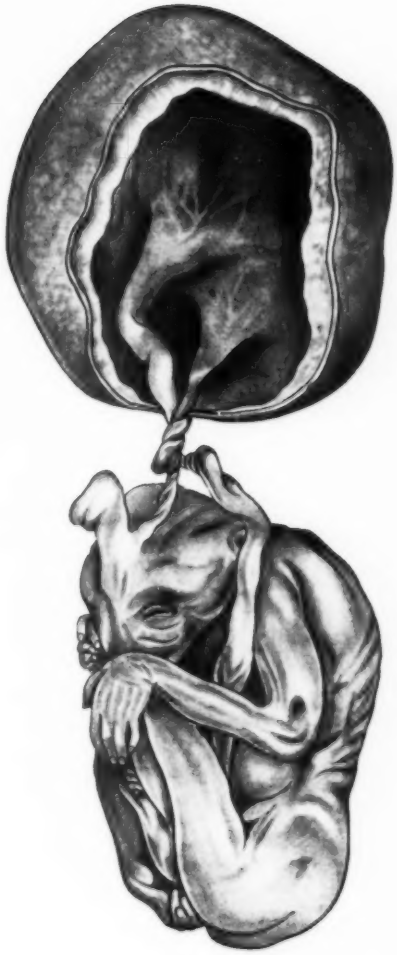

Fig. IV.

Fig. IIIb.

Fig. IIIa.

Fig. Ia, b.—Pero- et Syndactilia ligamentis amniotis effecta. Fœtus, 9 mens. Prag. Mus. 4943.
Fig. IIa, b.—Perodactylia ligamentis amniotis effecta. Female 70a. Prag. Mus. 5543.
Fig. III.—Ligamenta amniotica. Kind 3 hor. Prag. Mus. 5377.
Fig. IV.—Adhæsio amniotica interplacentam et caput fœtus papyracei. Prag. Mus. 5105.

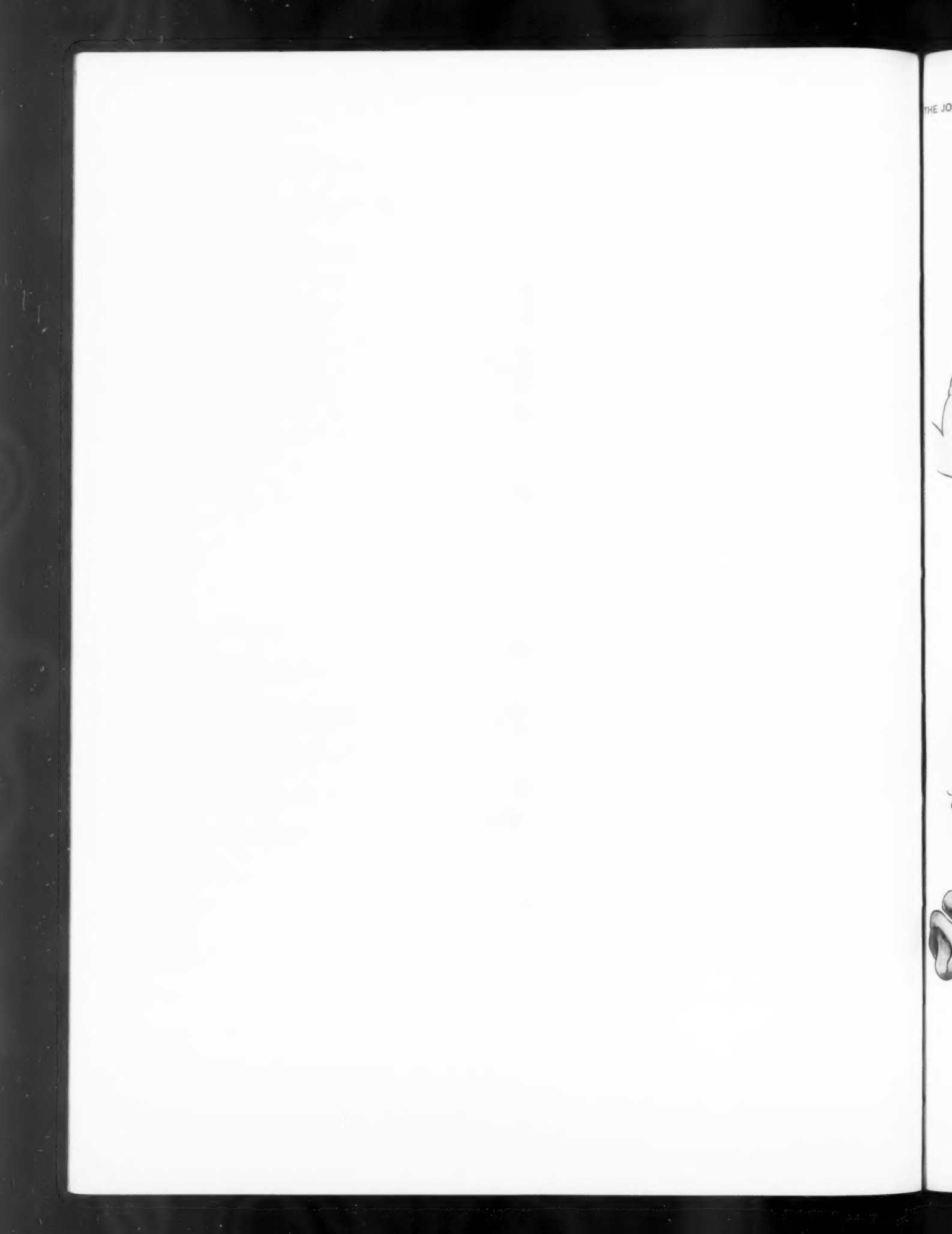

FIG. VIII.

FIG. VI.

FIG. V.

FIG. VIIa.

FIG. VIIb.

FIG. V.—Schizosoma cum defecta extr. s. d. Fœtus, 38 cm. Prag. Mus. 5610.
FIG. VI.—Enkephalokele. Prosoposchisis. Ligamenta amniotica. Strassburg. Mus. 1922.
FIG. VIIa, b.—Schizosoma. Prosoposchisis. Ligamenta amniotica. Involucrum amnioticum thoracis. Prag. Mus. 5490.
FIG. VIII.—Perobrachia sin. prob. amnio effecta. Male, 4 mens. Prag. Mus. 2417.

of the bone was arrested by the development of secondary exostoses about the bodies of the vertebræ.

CASE II.—Museum preparation 6628 (Figs. 3 and 4). This pelvis was found in a 55-year-old laborer whose corpse was drawn out of the Rhine, May 23, 1909. The body was very strongly built. The lumbo-sacral junction was very prominent, particularly on the right side, while the normal number of vertebræ were present in the cervical, dorsal and lumbar regions. On either side, the sacrum presented four foramina and the first coccygeal vertebra (the 30th), was typically caudal in character.

On inspecting the pelvis from behind, it was seen that the spinous process of the fifth lumbar vertebra projected 1 cm. more posteriorily than the corresponding processes of the other lumbar vertebra. The continuity of the interarticular portions of the last lumbar vertebra was broken on either side, exactly midway between the upper and lower articular processes, and their rounded margins gaped to the extent of 1 cm. The body of the fifth lumbar had advanced to the same extent over the upper surface of the body of the first sacral vertebra. Following the olisthesis, hyperostoses had developed upon the anterior and inferior margin of the body of the fifth lumbar, as well as upon the anterior and superior margin of the body of the first sacral vertebra. The hyperostoses were 1 cm. high on the right side, but somewhat lower on the left, and a prolongation of the last intervertebral cartilage extended between them. The bodies of the several sacral vertebræ were fused together in the usual manner, and the sacral canal appeared to be closed as far as down the middle of the 28th vertebra. The pelvic inclination was very slightly diminished, while the bones of the pelvis appeared powerfully developed and free from any sign of injury.

Pelvic measurements: Distance between iliac crests 25.7 cm.; between anterior superior spines 23 cm. and between superior posterior spines 6.5 cm. The superior strait presented the following measurements: Conjugata verå, 10.2 cm.; transverse diameter, 12.7 cm.; right oblique, 12.2 cm.; left oblique, 12 cm.; right sacro-cotyloid, 7.6 cm., and left sacro cotyloid diameter 7 cm. Plane of greatest pelvic dimensions: Conjugata, 12 cm.; transverse, 11.3 cm. Plane of least pelvic dimensions: Conjugata, 11.8 cm.; distance between ischial spines, 10.2 cm. Pelvic outlet: Distance between tubera ischii, 11 cm. On either side, the sacral portion of the innominate bone measured 6.7 cm.; the iliac portion 6 cm., and the pubic portion 7 cm., while the sacrum was 12 cm. in length and 11 cm. in width.

In this instance the predisposing cause for the spondylolisthesis was exactly the same as in Case I, and we likewise had to do with an undoubtedly congenital spondylolysis interarticularis of either side of the fifth lumbar vertebra, from which the olisthesis gradually developed. At the time of death the olisthesis was only slightly developed, but it would in all probability have increased in extent had the individual lived; since, in the absence of the formation of synostoses, no mechanism existed to check further slipping.

CASE III.—Museum specimen 6651 (Figs. 5, 6 and 7). This pelvis was obtained from a 55-year-old peasant who committed suicide by hanging, August 9, 1909. The history gave no intimation of the existence of any abnormality on the part of the vertebral column, and the skeleton was tolerably strongly built. On removing the viscera at autopsy, it was noticed that the region between the fourth and fifth lumbar vertebræ was more prominent than usual, and also that the left transverse process of the fifth lumbar vertebra was very wide.

On more careful examination, it was found that a slight olisthesis, to the extent of 1 cm. had occurred, so that the body of the fourth lumbar vertebra projected over the anterior margin of the fifth. This was made possible by a spondylolysis in both interarticular portions of the fourth lumbar vertebra. The lower margin of this vertebra was drawn out in a lip-like manner, more particularly in its anterior portion, which was united by ligamentous structures with a similar lip-like hyperostosis, about 1 cm. thick, arising from the anterior and upper margin of the body of the fifth lumbar vertebra.

The separation in continuity in the interarticular portions of the fourth lumbar vertebra lay somewhat more posteriorily on the left than on the right side. On the right side it extended between the lower and middle third of the joint surface of the upper articular process, while on the left side it extended only through the lower end of the upper articular process. An irregularly bowed bony process extended from the upper articular process on the right side and grasped like a claw the corresponding interarticular portion. The two fragments of the vertebral arch of the fourth vertebra lay about 1 cm. apart and the intervening space was filled with ligamentous structures.

As compared with the spinous processes of the third, second and first lumbar vertebræ, that of the fourth vertebra was very prominent. The fifth lumbar vertebra presented no lesion in continuity in the interarticular portions, but the inferior margin of its body, like the upper margin of the first sacral vertebra was somewhat hyperostosed. The right transverse process of the former appeared normal, but on the left side there was a bony plate about 3 cm. in diameter and concave below, which was firmly united by ligamentous structures with a correspondingly formed protuberance arising from the upper surface of the left wing of the first sacral vertebra.

The sacrum was made up of six vertebræ of which the sixth was formed by the 30th vertebra, which was definitely assimilated to the sacrum, below which came three fused caudal vertebræ. On the left side of the 29th and 30th vertebræ there was an irregularly formed, jagged exostosis, while the entire left half of the former appeared to be irregularly hyperostosed. Four sacral foramina were present on either side. The ilio-sacral joints were open and their anterior margins somewhat hyperostosed. The pelvic inclination appeared normal. The descending ramus of the right pubic bone presented a fracture, which had healed with a perceptible hump, while the ascending ramus of the left ischium likewise presented a fracture, which had healed by hyperostosis.

Pelvic measurements: Distance between crests, 23.5 cm.; between anterior superior spines, 20.5 cm.; between superior posterior spines, 7 cm. Superior strait: Conjugata vera, 9.3 cm.; transverse diameter, 15.5 cm.; right oblique, 11.2; left oblique, 11.2 cm.; right sacro-cotyloid, 9 cm.; left sacro-cotyloid, 9 cm. Plane of greatest dimensions: Antero-posterior diameter, 11.5 cm.; transverse, 10.5 cm. Plane of least pelvic dimensions: Antero-posterio diameter, 11.5 cm.; distance between ischial spines, 8 cm. Pelvic outlet: Distance between tubera ischii, 8 cm. On either side, the sacral portions of the innominate bone measured 7.5 cm.; iliac portions, 6 cm., and pubic portions, 6 cm. The sacrum was 12 cm. in height and 11 cm. in width.

In this specimen, the olisthesis of the fourth lumbar vertebra was likewise the result of a spondylolysis interarticularis. Leaving out of consideration the fact that the olisthesis involved the fourth lumbar vertebra, I consider that the interesting feature of the case is the probability that the spondylolysis interarticularis could be attributed to a fracture. This would seem to be borne out by the irregular outline of the separation of the interarticular portions, which on the right side extended through the superior articular process itself, and in addition by the presence of undoubted traces of fracture on the anterior wall of the pelvis and the lower end of the sacrum. The hyperostosis of the left transverse process of the fifth lumbar vertebra, as well as that of the left ala of the first sacral vertebra might also have resulted from a fracture, but I consider it more likely that they are secondary processes resulting from the olisthesis.

Therefore, I believe that this man had sustained a fracture of

the pelvis, involving the arch of the fourth lumbar vertebra, from which the spondylolisthesis had gradually developed, while fixation had not occurred up to the time of his death. It is greatly to be regretted that a history of the case is entirely lacking.

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ON DIABETIC ACIDOSIS.*

By Prof. A. Magnus-Levy, Berlin.

What do we understand by the term acidosis, a name first employed by Naunyn? Acidosis means the *accumulation* of acids in the body, in sharp distinction to the *formation* of acids which are produced, indeed, in every moment of normal life. In the normal metabolism substances of acid or alkaline properties are continuously being formed, but the tendency of metabolism is towards their neutralization. Probably all acids, which we encounter in pathological states, are produced as intermediary products in the normal change of matter. We know this to be true of uric acid, of oxalic acid and of glycuronic acid, etc.; we may assume it of homogentisic acid, of oxybutyric acid and of others. We, therefore, are not justified in defining acidosis, simply as a formation or as an appearance of acids which do not occur in normal health.

The accumulation of acids, the essential character of acidosis, presupposes in each case a disturbance of oxidation. Whether, besides the disturbed oxidation an increased formation of acids is present in acidosis is another question, and must be answered separately in each single case. (If homogentisic acid is a regular intermediary product in the katabolism of phenylamine and tyrosine, as some authors believe, its quantity will depend merely on the amount of katabolized albumen. The alcaptonturic individual will not produce larger quantities of this acid than a healthy person, but in the normal individual total combustion of the intermediary products takes place, while in the alcaptonuric patient the acid is neither split up nor oxidized.)

Similarly with oxybutyric acid, the principal subject of my lecture, we must consider whether it is formed in a larger amount in diabetes than in normal life.

Besides these that I have mentioned, other acids occur as results of metabolism, such as lactic acid, volatile fatty acids, higher fatty acids, amygdalic acid and carbonic acid. Some among these display specific effects, even when present only in small quantity, as does uric acid, oxalic acid and perhaps the lower fatty acids. These specific effects do not come under the term of acidosis, which simply means the general result of acidification. This, indeed, takes place only when acids accumulate in very large quantities, such as are sufficient to disturb by their acid character the processes taking place in the organism. If we leave out of question the end product of all oxidation, carbonic acid, which has scarcely the character of an acid, only one acid remains, to which we may apply the name of acidosis in the above sense. This acid is the oxybutyric acid and dependent upon it diacetic acid. Whether besides this group another kind of acidosis occurs in pathology, namely, an acidosis produced by lactic acid, as yet seems doubtful.

You are all aware of the fact that oxybutyric acid undergoes oxidation into diacetic acid, and that from the latter acetone can be formed. It is hardly necessary to speak at length of the latter body, which has been the subject for intensive re-

* Paper read before the Johns Hopkins Hospital Medical Society, May 9, 1910.

search for many years. Two authors, Folin and Embden, found simultaneously that the acetone present in the urine is not eliminated as such at all, or only to a small part. It is a product of self-disintegration of the diacetic acid transferred by the kidneys from the blood into the urine. The acetone, too, which is exhaled by the lungs, is not present in the blood as such, but occurs in the form of diacetic acid.

Whenever tests for acetone are positive, we should consider diacetic acid to be present. The name acetonuria, still in use, may be used as synonymous with acidosis. The strong and characteristic smell of acetone in the expired air is an excellent guide for its diagnosis. With a subtle sense of smell one is able to observe the presence of acidosis and even to differentiate its intensity. In my experience the smell of acetone is observed in almost every tenth patient entering the hospital. I would like to emphasize here that acidosis is a very common occurrence. Acetone bodies, which is a comprehensive name for oxybutyric acid, diacetic acid and acetone, are temporarily formed in almost all diseases. Several, even numerous, forms of acidosis or, as one called it formerly, acetonuria, have been described. But all these forms are identical when considered from an etiological standpoint and are all to be classified under the same head. In general, acetone bodies are eliminated always when carbohydrates are lacking in the organism, or, when they are not utilized in a normal way. Under these conditions, indeed, the oxidation of protein and of fat will be deficient and oxybutyric acid will appear in the urine. Acidosis will occur *every time*, when from any reason, the taking of food and especially of starch and of sugar, sinks to a minimum.

This applies to all kinds of acetonuria—to the acetonuria from starvation or from exclusive animal food, to the acetonuria in fever, in gastro-enteritis and to the acidosis appearing in the latter stages of cancer. Only upon one point do we still remain in doubt, whether besides the absence of carbohydrate, there exist *other* circumstances, which, although they do not induce acidosis, yet may increase and modify it. Practically this question is rather irrelevant, and I think it an unnecessary discussion of the subject. Another question is of far greater importance, and that is the difference between diabetic and non-diabetic acidosis. Except in diabetes the elimination of acetone bodies rarely exceeds 10 or 15 grams, and when such quantities appear in urine, they do so only for a short time; in diabetes, on the contrary, the elimination may reach 30, 40 and more grams, and acidosis of this height may last for months and even for years. We know this fact since the early eighties, a period when we did not yet employ alkalies.

Here I would like to call your attention to the very important deduction, that a patient with severe diabetes, and with a very marked acidosis, may, under favorable circumstances, live for years without suffering *immediate* damage to his health. He is, it is true, according to Frerich's words, menaced by the danger of a certain and unexpected death, but this end may not be reached for many years. And this danger is imminent only in the *diabetic* patient, whose capacity for utilizing carbohydrates is extremely weakened. When the interest in acidosis became general among practitioners, when the Legal-Gerhardt test, so often positive, revealed the frequency of strong acetonuria in non-diabetic diseases, a feeling of apprehension arose. The bad prognosis which persistent and marked acidosis gives in diabetes was transferred without reason to acidosis existing in diseases other than diabetes. But acidosis never is a threatening danger in *such* patients. Acidosis as such has nothing to do with the symptoms and with the dangers of such diseases. The idea of the specialists in children's diseases was wrong, who, in studying the acidosis which occurs in the alimentary disturbances of nursing babies, ascribed the cachexia and death of the children to oxybutyric acid.

Furthermore, in diabetes acidosis, even when it has lasted for years, induces no *immediate* harmful consequences. Thus acidosis, which exists other than in this disease, being far weaker and of short duration, is without clinical interest and without prognostic value.

You are aware that the human organism, as well as that of carnivorous animals, defends itself against the action of accumulated acids by forming an alkaline body, ammonia. A diabetic patient at any time has at his disposal 6 or 8 grams of ammonia. The neutralization of acids by ammonia prevents deprivation of alkalies and a change in the alkalinity of the most important cells. With 6 or 8 grams of ammonia, 40 or 50 grams of oxybutyric acid can be eliminated month after month, without acidosis becoming deleterious. A *fatal acid-poisoning* will occur only then, when the accumulation of the acids reaches twice or three times these amounts. There is a difference between *acidosis*, spoken of in the common sense, and a *fatal* acid-poisoning, a difference which though only in the quantity of acids, yet cannot be emphasized strongly enough. Outside of diabetes, such an enormous rise of the accumulation of acid never takes place, so far as we know at present. The coma produced by it is limited to diabetes and merits the name of coma diabeticum.

Stadelmann, a pupil of Naunyn, was the first, as you well know, to consider diabetic coma to be a fatal acid poisoning. He compared it to the experimental poisoning by muriatic acid in experiments with animals. According to Walther's fine discovery, rabbits, after having been given 0.9 gram of HCl per kilo of body weight within 24 hours, die in a comatose state. The striking analogy between this form of acid poisoning and that of diabetic coma, emphasized by Stadelmann, exists in the similarity of the clinical symptoms, especially the changed and deepened breathing, further in the strongly diminished quantity of carbonic acid of the venous blood, together with the diminution of the alkalies as determined by titration. Aside from these symptoms the occurrence of ammonia in the urine is common also to both states. One link alone was missing in the chain of proofs furnished by Stadelmann. It had not yet been demonstrated that such an amount of acid, as was necessary to bring about the death of the rabbit, was present in a case of human diabetes. The *urine* of comatose diabetic subjects not only contained no larger amount of organic acid, but rather less than in the time pre-

ceding the coma. With one exception this quantity did not exceed 20 grams. That is the reason the theory of Stadelmann was doubted. The total quantity of acid found in any given case *appeared* insufficient.

A mistake had been made, however, in looking for their presence in the *urine* of deceased patients; it would have been just as much of an error to have looked for muriatic acid in the urine of dying rabbits. The lessened alkalinity of the kidneys diminishes the secretory power, the stream of urine decreases rapidly, sufficient elimination of the acid either ingested in an experiment, or formed within the body of a diabetic patient does not take place. Both the poisoned animal and the diabetic patient do *not* die from the acid which has been *eliminated* in a neutralized state, but from the acid which *remains in the body*. The presence of the acids was not to be looked for in the *urine*, but in the *organs* of the corpse. By laying stress upon this point of view I really have been able to find in the body by analysis of blood, liver, muscles, etc., quantities of 100 to 200 grams of oxybutyric acid. Calculated per kilogram and allowing for differences in molecular weight, these amounts are just as large, and even larger, than those which induced the death of Walther's rabbits.

In the urine also enormous quantities may be found, but only if the patient survives the comatose state, so that the elimination of acids takes place in a sufficient way. I was lucky enough to establish this fact in a boy under the care of my teacher, Naunyn. This boy, only 30 kilograms in weight, underwent a most severe coma, but was saved by the use of almost 200 grams of sodium bicarbonate in 24 hours. On this day the urine contained 160 grams of acetone bodies; the following days the elimination sank to a lower level.

In the experimental poisoning by acid, there exists an *experimentum crucis*, which proves the death to be really due to the action of the acid itself and not to secondary factors. If the acid circulating in the body is neutralized by intravenous injection of bicarbonate of soda, improvement follows immediately even when death seems to be imminent in the very next minutes. This *experimentum crucis* has succeeded also in diabetic coma, though only seldom, just as it was postulated by Stadelmann. I myself have seen three cases survive pronounced coma by use of bicarbonate of soda, two of them being children, one a patient of sixty years.

Here in America the doctrine of Stadelmann-Naunyn was well received and also it has been developed farther. In Germany, too, it has been acknowledged more than formerly since the new proofs have been furnished. According to my personal opinion, scarcely any other theory upon the entire field of metabolic science has better support and has been worked out more thoroughly both qualitatively and quantitatively. Yet it is proper that all objections which have arisen against it in the course of years should be considered carefully.

Four objections are to be named. The first one is the relative inefficiency of the alkaline treatment. It must be conceded that only few cases are known, in which an advanced diabetic coma has disappeared under the use of bicarbonate of soda, in contrast to the *absolute* efficiency of alkalies in the case of the rabbit. But it must not be forgotten that there is a marked difference between acid poisoning in experiments and in coma. In the first a certain limited and well-known amount of acid is introduced into the organism; when this quantity has been neutralized by the once-administered bicarbonate of soda, danger is over at once and will not reappear. During diabetic coma, on the other hand, the formation of acid does *not stop a moment;* new molecules enter continuously into the metabolism, to such a degree that the ingestion of bicarbonate of soda does not keep step. In order to neutralize 150 grams of oxybutyric acid, formed in the course of 24 hours, a quantity of 120 grams of bicarbonate of soda is required. Even when absorbed, the arrival of the alkali at the menaced centers of the brain and its utilization there is by no means to be taken for granted in every case. Only when the immense production of acids diminishes, or rather if oxidation takes place again—which, however, is not the case, as long as coma is at its height—will the alkalies ingested be sufficient to neutralize the acids. The urine then becomes alkaline and the diabetic patient will survive the coma. This quantitative calculation reveals why bicarbonate of soda therapy fails so often in advanced coma, but at the same time it is obvious that this failure does not contradict the theory of Stadelmann.

The second objection to be mentioned seems to be of more importance. It arose from the modern theories of acidity and alkalinity. Since acidity and alkalinity have been defined by the teachings of physical chemistry as the degree of concentration in hydrogen and hydroxyl ions, it has been pointed out that the fluids and the secretions of the body are fairly neutral, if measured with this measure, with the single exception of the gastric and pancreatic secretions. The amount of hydroxyl and hydrogen ions in the blood serum does not undergo marked variations either in the various conditions of normal life or in illness. Even in the acid-poisoning of rabbits only a slight derangement of the neutral point of the reaction occurs and a slight decrease in the alkalinity takes place. In diabetic coma, however, such a decrease has not been found. Silli and Benedict, who carried out these experiments, therefore, deny any analogy between diabetic coma and the experimental acid-poisoning. They deny that the former comes under the head of acid-poisoning.

But their argument does not hold good. Besides other objections, which could be made, the main difference lies in the fact that Benedict, unlike Walther, did not inject the muriatic acid into the *stomach* of the animals, but introduced it in the form of a continuous stream into the veins. The blood was mixed with the acid fluid directly, without the latter having been passed through the walls of the intestines, and the intravenous injection was continued until death. Under these circumstances the actual concentration of ions at the moment of death, of course, is higher than that, which would have been reached if the acid had entered slowly and evenly from the stomach into the blood. I think, if the acid-poisoning had been produced in the same manner, as in Walther's experiments, the alkalinity of the ions would have been the same as in diabetic coma; in other words, that here also the quantity

of hydroxyl ions would not have been distinctly diminished. Apart from these objections there is still another difference between experimental poisoning and diabetic coma. In the example of the rabbit the acid enters first the blood and only after that the cells; in the diabetic patient, on the contrary, the acids are actually produced in the cells themselves, and only a fraction of the total quantity enters the blood. Thus with the same degree of change of alkalinity in the nerve centers, upon which depends the death of the organism, the decrease of alkalinity of the blood will be greater in the animal than in the human patient. Death in both is not to be considered simply as a consequence of the changed reaction of the *blood*, but in reality as due to the poisoning of the *vital center*, by which in particular are meant especially the respiratory centers.

It is to be regretted, that we are still unable to measure the alkalinity of the *organs* themselves. But in spite of this lack we have complete evidence of the influence of acids upon what is taking place in the organism. I will give you a striking example from the field of normal metabolism. The production of even small quantities of lactic acid in the working muscles, and their entrance into the blood will excite the breathing center, although in this case, on account of the *small* amount of acids brought into play, no change in the ionic concentration in the blood occurs. This excitement of the respiratory centers is to be compared with the far stronger irritation occurring in coma. I will remind you also of the dreaded casts in the urine, which suddenly appear in many cases before the outbreak of the coma. I believe them to be acid casts, and I think that they indicate a local decrease of alkalinity in the kidneys, whilst the alkalinity of the blood and of the vital brain centers is yet normal.

To sum up the objections regarding the state of reaction in the organism I would say that alkalinity, in the sense of physical chemistry, and alkalinity in the common language of chemistry and of biology, are by no means identical. When there is no change in the ionic concentration, lessened alkalinity in the biological sense may be present and may be of greatest importance.

Two older objections to Stadelmann's theory are mentioned to-day more rarely than in former times, but even to these consideration must be paid. Sometimes a comatose state was encountered in diabetic patients, who did not show any signs of true acidosis. Neither the deepened breathing was present nor was the amount of carbonic acid in the blood diminished; the analysis of the urine failed to give oxybutyric acid. Upon closer observation of the clinical symptoms *these* comatose attacks have proved to be phenomena of a different order. A diabetic may die quite as well of uræmic coma or of epileptic coma, and even the weakness of the heart may be accompanied by a sort of coma. These forms of coma have nothing in common with the dyspnoic form of coma save unconsciousness. The critical eye of the clinician has little difficulty in recognizing them. Objections of this kind seldom are met with at present.

On the other hand, there have been observed symptoms of dyspneic coma in non-diabetic diseases. I have seen myself such a dyspneic coma only once and then in a uræmic patient;* but older authors of the eighties affirm that it was by no means exceptional in *uræmia* and in *fever*. Here also a diminution of carbonic acid in the blood and of the titrated alkali occurs similar to that in diabetes. Since the comatose state in nephritis and diabetes have at least two symptoms in common, must we consider that acid-poisoning is present also in uræmia? And is fatal acid-poisoning a symptom which is not specific for diabetes? Also in infectious diseases similar changes of the alkalinity are held to have been found. Of all the objections against the doctrine of Stadelmann, I believe *this one* to be the most important. A full contradiction and definite refutation of this objection—I own it frankly—is as yet not possible; no detailed description of the clinical symptoms is given in the older observations which have been published, and the method of measuring the alkalinity has generally been inadequate. No attention has been paid any more to such events for 20 years, and I myself, though highly interested in the subject, have not had occasion to make researches upon this point, either clinical or experimental. But I am inclined to believe that a certain kind of *weak* acidosis could be in play here too. It is conceivable that lactic acid may have accumulated within the body of uræmic or infected patients. New inquiries on this point are needed. It should also be determined, whether acids are stored up in the tissues of such patients in the same way as in diabetic coma. Although one should be cautious in predicting the results of future investigations, I will express my opinion that neither of these lines of research will reveal the existence of a true acidosis in nephritis, *fully* corresponding to diabetic acidosis. Even should an accumulation of lactic acid be found, there will be a difference in its intensity. Although certain symptoms of acidosis as lessened alkalinity of the blood and deepened breathing may be present, other and more important symptoms may be missed, and also with the first symptoms differences are to be found. I find a support for this opinion in an unpublished piece of work of Fraenkel, a pupil of Kraus. He demonstrated that in spite of a certain resemblance, the figures of alkalinity obtained either by titrimetric methods or by measuring the carbonic acid in uræmia and infectious diseases were by no means identical with those obtained in the diabetic coma.

Having thus discussed the clinical aspect of acidosis I turn towards the chemical problem.

The inquiries upon the chemical connections of the acetone bodies, performed during the last decade, have broadened our knowledge, not only regarding the special pathogenesis of diabetic acidosis, but even concerning the general problems of metabolism.

It has been well known for years that acetone and the allied acids do not originate from carbohydrates. In the old controversy, however, the first reliable proof as to whether pro-

* Having just returned from America I observed it for a second time in a uræmic patient, but this time on closer observation, its character struck me as being different from the dyspneic coma in diabetes.

tein or fat was the mother-substance of the acetone bodies, was given only then, when it became evident that in a single experiment the amount of metabolized protein did not suffice for the formation of oxybutyric acid. I found in a boy suffering from diabetic coma, during three days a sum total of 342 grams of acetone bodies in the urine, but during this space of time only 270 grams of albumen were decomposed, in other words a far smaller amount. From the protein large quantities of sugar had been formed; it was, therefore, obvious that the protein alone could not have furnished such immense quantities of organic acids in the same time. In this example at least a great part of the acetone bodies must have been derived from the fat. However, this fact did not contradict a simultaneous formation from protein. Further experiments, carried out by Schwarz, Embden and Baer, proved, indeed, that acetone bodies may originate from both protein and fat.

All the researches of these authors are based upon the principle of feeding—in the presence of acidosis—a certain substance and of observing its influence upon the output of acetone bodies. Previous experiments had given no clear results, because the unbroken molecule of protein or fat had been given. Schwarz on feeding butyric acid was the first to examine the influence of *intermediary* products of fat. Later on amino-acids, the components of the protein molecule, and different fatty acids which could be assumed to be intermediary products of the katabolism of fat were administered in such experiments. Such investigations were necessarily carried out, as I said before, when acidosis of a certain degree was present, that is upon diabetic patients or dogs, or upon healthy animals and men fed exclusively on animal food. However, as spontaneous variations occur frequently in acidosis, the results of such feeding experiments are somewhat doubtful. A discovery of Embden, therefore, was of great advantage in the study. Embden, as you know, found that the isolated dog's liver on artificial circulation produced diacetic acid, which was no doubt derived from the oxybutyric acid. The amount of acetone bodies transferred from the liver to the blood increased considerably when certain substances were added to the blood. This method of investigation, employed with great skilfulness by Embden, has proved its value by giving striking results. Both methods of inquiry, that on the liver and that on living diabetic subjects, supplement and control each other exceedingly well.

We learn from them the following facts: The immediate predecessor of oxybutyric acid is *butyric* acid, which, by oxidation in β position, is converted into its oxy-acid. Butyric acid has shown itself to be the strongest producer of acetone bodies. The reason is to be found in the fact that this conversion needs only a simple and slight chemical reaction, the addition of one atom of oxygen. The higher fatty acids, however, have to undergo far greater decomposition, a splitting off of many atoms of carbon, before they reach the stage of a chain with 4 C atoms. In feeding fatty acids, with from 4 to 10 atoms of C, it became manifest that capronic, caprinic and caprylic acids, all having an even number of carbon atoms yielded oxybutyric acid, while other acids with an uneven number of carbon atoms, such as propionic and valerianic acids, did not yield oxybutyric acid.

Inasmuch as true fatty acids with an uneven number of carbon atoms occur but rarely in the animal metabolism, or only in small quantities, the practical value of this demonstration is of no importance in the metabolic changes in diabetes. For biological chemistry, however, this discovery was of great moment. From it, in connection with other experiments, we have been enabled to conclude that the katabolism of fatty acids goes on by the loss of two carbon atoms from the original chain. The splitting off begins with an oxidation at the β carbon atom. Thus a β oxy- and further on a β keto-fatty acid is formed; finally the β C atom is found to be at the end of a new fatty acid in the shape of a carboxyl group. In this way from stearic acid very likely is formed palmitic acid, and from this an acid with 14, 12, 10 and 8 C atoms is derived, and then capronic and butyric acid.

In the same way as acid decomposition goes on by the loss of a chain of two carbon atoms, so the building up of fatty acids by synthesis seems to be accomplished by a gradual apposition of two combined carbon atoms. The formation of fat, or rather of fatty acids from sugar, which in the animal kingdom goes on in large proportions, has been explained in this way by Nencki, Hoppe-Seyler and myself, and Heath and others have found new supports for this theory.

The relations existing between amino-acids and acetone bodies are not so clearly known as with the fatty acids because the chemical processes are more complicated. Some of the amino-acids, leucine, tyrosine and phenylamine, seem to be katabolized to oxybutyric acid, while others, alanine, valine and glycocoll apparently are not, but appear, on the contrary, to diminish the output of acetone bodies. In studying these substances very interesting results have been obtained regarding the oxidative decomposition of the amino-acids which up to this time has been completely unknown.

However, I will forego a discussion of the purely chemical aspect of this question. For the present it is sufficient to state the principal result of these investigations, which is a well-marked antagonism between two groups of amino-acids. Protein is by no means to be considered as a unit, in so far as acidosis is concerned, because some of its components may *increase*, and others *decrease* acidosis. So, too, in the formation of sugar the two groups behave differently. It should be remarked that those amino-acids, which in the present state of our knowledge are convertible into sugar, do not yield oxybutyric acid, while on the other hand that group of amino-acids which undergo transformation into acetone bodies does not give any sugar. Since the protein molecule contains groups of different behavior regarding acidosis, it is clear why it failed to show obvious results in those experiments, in which the whole unsplit protein molecule was given.

Ten years ago I mentioned another possible way for the formation of oxybutyric acid. I thought that it might be formed in a synthetical way. This might take place by union

of two chains with two C atoms, but this way of formation has not yet been proven. Under this conception alcohol could furnish the material for the synthesis, but not only does it *not* increase the amount of acetone bodies in urine, but, on the contrary, it rather seems to diminish them.

Allow me to cast a retrospective glance at a period in which therapeutists have drawn erroneous conclusions from the results of these inquiries. When at the end of the last century only the fat, but not yet the protein, had been recognized as a source of acetone bodies, some physicians thought it necessary, to restrict the amount of fat taken by the diabetic, because they feared to raise the acidosis by giving fat. There are two errors in this deduction. The first is the wrong supposition, that increased *ingestion* of fat means increased *decomposition* of fat. But any one, possessing an elementary knowledge of the metabolism of matter and energy, knows that this is not the case. A surplus of fat given in the diet does not raise oxidation any more than does a higher amount of oxygen in the respired air. There is no danger that by *giving* more fat more fat will be decomposed, and more acetone bodies set free in the body. And even if this were true, a second factor was neglected, the factor of the destruction of the formed oxybutyric acid. Acidosis, and I repeat it again and again, does not only, or perhaps not at all, depend upon the *formation* of acids, but also and perhaps exclusively upon their combustion. Indeed after analyzing and taking into account the results of all experiments carried out in this direction, I may say, that the ingestion of fat in almost no case of diabetes has induced a marked increase in the output of acetone. An exception is given alone by butter, on account of its richness in glyceryls of butyric acid.

In opposition to the behavior of fat a higher intake of protein may sometimes increase acidosis. But this does not happen in a direct way, due to the formation of oxybutyric acid from protein, but in an indirect way. The demand made upon the katabolizing and oxidizing powers of the organism by large quantities of protein ingested, may lessen the oxidative powers which concern the combustion of acetone bodies. Such disturbances may arise also from other incidents such as troubles of the digestive tracts. Since fat does not *directly* increase acidosis, we should always try, not only to maintain the weight of the diabetic patient by a sufficient supply of fat in the diet, but even to increase the same.

The endeavor to avoid those food stuffs, which may *form* oxybutyric acid, is in vain, since protein and fat each do so. Alcohol and sugar—and the latter is not available by the diabetic patient—are the only material which do not yield acetone bodies. We ought rather to look for some substance which would diminish acidosis by increasing the splitting up and the combustion of the acetone bodies.

Numerous substances are known which do this, and they are included under the name of anti-ketonuric substances. The carbohydrates exert the strongest anti-ketonuric effect. From 50 to 80 grams of sugar are sufficient in a healthy man to dissipate a strong acidosis, induced by a starvation or by exclusive animal food. As yet we do not know how this effect is accomplished. Naunyn's explanation that the decomposition of the acetone bodies is due to the simultaneous combustion of carbohydrates by secondary oxidation is merely a paraphrase. We are, therefore, compelled to look upon the occurrence as a fact without explaining it. A like efficiency is due to the preformed carbohydrates of the food, and to those which are newly formed within the body from protein or other material. *Glycerine* owes its anti-ketonuric efficiency to its being transformed into sugar. In healthy men the decomposition of large quantities of albumen can dissipate acidosis. Its efficiency likewise is to be referred to the partial conversion into sugar. For those very amino-acids—alanine, glycocoll and aspartic acid, which in the experiment of Embden and Baer showed an anti-ketonuric effect—are the ones from which sugar is formed. However, glycerine and albumen, which in healthy persons diminish the quantity of acetone bodies, are as good as useless in severe diabetes.

This fact, which previously appeared surprising, is now understood. The sugar, formed from glycerine and from protein, whose combustion induced the combustion of acetone bodies in *healthy* persons, is eliminated in severe diabetes and thus cannot be utilized for the oxidation of the acetone bodies.

Most other materials, known to be anti-ketonuric, such as gluconic acid and others, are not food stuffs and, therefore, cannot be employed permanently. Alcohol, though in bad repute as a poisonous substance, is the only food stuff, which acts beneficially on the diabetic acidosis. It has been shown by experiments of Neubauer and of Benedict that it decreases materially the amount of acetone bodies in the urine. In coma I would not only consider the ingestion of alcohol to be allowed, but even to be desired. Therapeutic experiments with this substance in coma require great discretion because of the effect upon the patient and judgment in the interpretation of this effect. Besides alcohol no other compound of the food is at our disposal for combating acidosis. The difficulties of the struggle against acidosis depend upon the fact that the energy of sugar, the most powerful anti-ketonuric body, is not available in severe diabetes. Every improvement in the toleration for carbohydrates will be followed by a decrease in acidosis; but the general treatment of diabetes is outside of the limits of my lecture. You may attain your purpose of increasing sugar combustion and at the same time lessening acidosis by different means: you may restrict the quantity of protein in the food, you may prescribe fasting and vegetable days, you may give an oatmeal diet for two or three days, you may prescribe muscular exercise, or you may combine all these measures, and this will depend on the peculiarities of each individual case.

The use of bicarbonate of soda, which we continually employ in the treatment of severe acidosis, does not act directly against it; neither does it diminish the formation of acetone bodies, nor does it favor their combustion. In many cases, on the contrary, the amount of acetone bodies found in the urine

will increase, if great quantities of alkali are given. That by no means signifies a weakness of the oxidative powers, nor must it be considered as disadvantageous, or even dangerous. Rather the contrary is the case. While in general the amount of organic acids in the urine may be considered as an indicator of the degree of acidosis, this is different when alkalies are administered. Alkalies facilitate the elimination of acids by neutralizing them and thus withdraw the acids from oxidation; thus the amount of acids present in the body in a certain moment is diminished, and sufficient alkali is present always to neutralize them and to prevent poisonous action upon the cells. Naunyn contends that he has never seen a sudden death by coma following the withdrawal of carbohydrates since the time when he began to give sufficient quantities of alkalies from the beginning of a strict diet. But no matter how beneficial is the action of alkalies, we should not forget that they act only as a palliative remedy and that we should seek for more efficacious substances with which to combat directly the acidosis.

If that substance is once found, which will enable the diabetic to utilize carbohydrates, acidosis and its dangers will have disappeared.

I return to the problem which I touched slightly at the beginning of my lecture. I have mentioned the possibility that acidosis might have nothing to do with an increased formation of oxybutyric acid, but that it depended exclusively on disturbances of oxidation. One may imagine that also in normal life oxybutyric acid may be formed *to the same extent* as in the coma. This doctrine presupposes that each molecule of fatty acid and of amino-acid, which, according to our knowledge *may* be transformed into acetone bodies *must* really be so without exception. In other words, that this way of decomposition of fats and certain amino-acids, leading to oxybutyric acid, is not a facultative one, but that it is *obligatory*. I was able to prove that in diabetic coma such a maximum of oxybutyric acid was formed. One of my patients had decomposed 90 grams of protein and almost 200 grams of fat within 24 hours. From these substances, as far as calculation is possible to-day, about 110 grams of acetone bodies could be derived. This quantity really was found to be eliminated by the urine during three subsequent days. A healthy man living on a carbohydrate free diet would consume about 120 grams of albumen and 210 grams of fat daily; from these about 120 grams of oxybutyric acid could be formed; that means about 1½ to 2 grams per kilo. Are we allowed to assume that he really forms such quantities of acids? We are allowed to do so if we can make it probable, that he is able to destroy them by combustion. Rabbits and dogs have oxidized 3 grams of this acid and even more per kilo introduced at one time into their stomachs. Since combustion is far easier when such substances are formed within the body, it might be possible for a healthy man to decompose 2 grams per kilo.

I furthermore call attention to the great amount of acetone bodies, which may be formed by non-diabetic organs. In Embden's experiments, the isolated dog's liver, an organ of scarcely 200 grams in weight, yielded 3, 4 and even 9 grams of diacetic acid, calculated for 24 hours. Since a part of it certainly had been destroyed, in the course of the experiment the production was in reality much larger.

No reasons exist in my opinion to disprove the conception that a healthy man may form and oxidize such large quantities of acetone bodies. For a long time these substances have been recognized as products of intermediary metabolism. According to my doctrine they would not be *facultative*, but they would be the *obligatory* products of the decomposition of fatty acids and of some amino-acids. If this doctrine is right, you see clearly that acidosis is *only* the consequence of disturbed oxidation. However, this conception is as yet not absolutely proven.

Embden takes the opposite standpoint. He observed that the surviving dog's liver, which was found to *produce* acetone bodies, was able also to *katabolize* diacetic acid. In continuing his researches on the liver of a depancreatized dog, he found that the destruction of diacetic acid was not smaller than with the liver of a healthy animal. Thus finding no decrease of oxidation, he considered acidosis to be caused by increased formation of acids. However, it is not allowable to transfer the results of the experiment upon a dog to the metabolism of man. Both behave differently. In living dogs accumulation of acids does not occur so generally and to the same intensity as in man, they do not lose the capacity for combustion of acetone bodies so completely as diabetic patients. There is a very marked difference between carnivorous and omnivorous animals. Men, using carbohydrates as food, destroy the oxybutyric acid, formed from fat and from albumen, *only* by the help of carbohydrates; they lose this capacity more or less if they become unable to katabolize the sugar molecule. Carnivorous animals, on the contrary, living on meat and fat, are enabled by nature to burn oxybutyric acid *without* the help of ingested carbohydrates. That means a wise arrangement of nature—and thus in diabetes of dogs accumulation of acids never reaches such an extent as in men.

You might ask me why accumulation of acids is increased so suddenly in diabetic coma. One may ascribe this sudden increase, at least in some cases, to a sudden decrease or even to a loss of the sugar combustion. Spitzer reports a case where this was obvious. Coma set in immediately after a strong physical shock combined with a psychical emotion. From that moment the elimination of sugar, up to then rather slight, rose to an abnormal height, and at the same time with increased loss of sugar the oxidation of acetone bodies was reduced to a minimum. Here no time was left for adaptation, which in the general course of diabetes prevents the rapid increase of acidosis. I might quote other examples where such a sudden increase of the toleration for sugar can be observed; I mean those very rare cases of acute diabetes, the beginning of which may sometimes be determined almost up to the hour by the sudden appearance of a terrible thirst in patients who until then showed not the slightest symptoms of diabetes. In such cases acidosis also arises suddenly and advances rapidly. The time being far advanced I confine myself to these few remarks.

It will not be quite easy to clear up all problems of this matter.

I am at the end of my lecture. I have not considered it my object to dwell upon those symptoms of acidosis which are considered more or less absolute facts. This was unnecessary with an audience so well informed on the subject as an audience of American physicians. I have undertaken the more valuable task to show, which problems are awaiting yet their answer and what objections may be raised to the doctrine of acidosis. To many of our profession the theme of acidosis seems to furnish no problems for future research. I hope to have demonstrated in which direction further research may be successful in order to advance both the theory of acidosis as well as its treatment.

SOME OBSERVATIONS ON THE BACTERIOLOGY OF THE BALTIMORE CITY WATER.*

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The importance of a thorough knowledge of the water used by the city of Baltimore cannot be overestimated, especially at the present time when heavy expenditures are to be undertaken for the enlargement and permanent improvement of the supply. It is essential that the character of the water collected in the various water sheds which feed the city reservoirs should be well understood, particularly its content in bacteria, and the extent to which it is polluted by microörganisms derived from the intestinal tract. The results of such a study belong essentially to the medical public of the city, and if these results indicate a pollution of the supply which is at all likely to be a menace to the public health, it is the duty of every one conversant with the bacteriology of the city water to make his knowledge common knowledge. The number and character of the microörganisms found in the water delivered to the consumer will determine in a great measure the necessity of filtering it through sand filters, unless a careful examination of the various tributaries of the main streams utilized by the city should reveal that the pollution which may be found has its source in a few small streams which can be eliminated from the system.

During the past six years I have been making regular bacteriological examinations of the water drawn from the tap in the Bacteriological Laboratory, and in the organization of a class in Practical Hygiene have had the opportunity of paying frequent visits to the valley of the Gunpowder River and of studying several of its most important branches in Baltimore County, particularly the Beaver Dam Run, the Western Run, and the Oregon Branch. For a number of miles in the vicinity of these streams the general conditions prevalent along their banks have been carefully scrutinized, especially any local conditions which might favor a sewage contamination. The water from our tap has its source primarily in the Gunpowder River, and it has thus been possible to compare the bacteriological findings of the water as delivered with those of various samples collected from the river itself and from the above-mentioned tributaries. Recently it has seemed wise to review the work of this fairly long period of time and the results found during the different years agree so completely as to make deductions from the evidence presented of considerable value. The main conclusions obtained do not differ to any appreciable extent from those reached by Dr. W. R. Stokes and his co-workers in the Bacteriological Laboratory of the City Board of Health, but in a question so vital to the well-being of a community as its water supply, it is of paramount importance that the work done in different laboratories should be carefully compared. Only in this way can observations of permanent utility be made.

TAP WATER.

The water supplied to the Bacteriological Laboratory is derived from Guilford Lake and thus from the Gunpowder River at Lock Raven. This water has been examined at frequent intervals by the simplest methods in vogue for the study of water bacteria. The colonies have been counted on agar and gelatin plates, with 1 cc., 1/10 cc. and 1/100 cc. quantities. The average of these three counts has been taken to represent the bacterial content of the samples. Fermentation tubes of 1 per cent dextrose broth have been planted with 1 cc. and 1/10 cc. quantities and rarely with 1/100 cc. When these tubes showed positive fermentation by the production of acid and the evolution of gas, plates have been poured from the open bulb, the various colonies fished and planted on agar, and subcultures made in agar, gelatin, potato, Dunham's solution, litmus milk and various sugars. These reactions together with the morphology and motility of the organisms have served to establish the identification of the various species.

The number of organisms per cubic centimeter in our tap water is not large. It is uniformly below the standard of purity, 1000 to the cubic centimeter, and is usually between 500 and 600. It may be as low as 300 to the cubic centimeter. On rare occasions it may be increased to 1500 or even 2000. A high bacterial count is not common and is usually associated with heavy rains in the vicinity of Baltimore and is accompanied by a great increase in turbidity. From the bacterial count alone there is little or no evidence of pollution. Fermentation tubes, however, show almost invariably a positive presumptive test for *Bacillus coli*. This is practically always

* Read before the Baltimore City Medical Society, May, 1910.

given in 1 cc. quantities and it has proved a rare event, during six years of routine examinations, for this test to fail. Occasionally when a single Smith tube is planted with such a quantity, there is no active fermentation of the sugar as a result, but if half a dozen tubes be planted at the same time the majority always shows a positive presumptive test. Not infrequently the tubes planted with 1/10 cc. also give positive results and on very rare occasions I have seen acid and gas develop in the tubes planted with 1/100 cc. When plates are poured from the fermentation tubes and the various bacteria isolated which give rise to the splitting up of the sugar, considerable diversity is evident in the characteristics of the microörganisms encountered. The principal isolations, nevertheless, have certain cultural features which indicate that their source of origin lies in the intestinal contents. The species may be grouped about six main types.

Group I. Bacillus Coli Type.

In the majority of instances the microörganisms found in the fermentation tube conform to the classical type of *Bacillus coli*. They are small, sluggishly motile bacilli which produce a rather thick, moist, non-pigmented growth on agar, fail to liquefy gelatin, acidify and coagulate litmus milk without digestion of the casein, and ferment dextrose with acid production and the evolution of a gas composed of a mixture of hydrogen and carbon dioxide, with the formula $\frac{H}{CO} = \frac{3-4}{1}$. Many of the strains isolated produce indol abundantly, the reaction being brought out by the addition of nitrites to the mixture of sulphuric acid and a Dunham's solution culture, and in many instances the usual dirty brown growth on potato is also seen. Neither indol production nor abundant growth on potato have been considered essential in the identification of the organism, the previously mentioned reactions being regarded as diagnostic.

Group II. Bacillus Proteus Vulgaris Type.

Organisms of the "proteus vulgaris" type have been isolated on a number of occasions. In morphology they are not unlike *Bacillus coli*, but are somewhat longer and thinner. They have a rapid darting motility characteristic of this group. Their colonies on agar and gelatin have spreading, *leaf-like* edges and the lattter medium is rapidly liquefied. On the surface of slant agar the growth is spreading and in stab gelatin cultures liquefaction progresses rapidly in a transverse direction along the surface of the solid medium. The organisms ferment dextrose with a gas formula of $\frac{H}{CO_2} = \frac{3-4}{1}$, the hydrogen being in excess of the carbon dioxide. Milk is not firmly coagulated but shows a soft clot, the reaction remaining alkaline, and the litmus being completely reduced. The clot is slowly dissolved and the litmus milk is eventually converted into a glairy, dirty brown fluid in which no trace of curd is visible. If fresh litmus be added, the reaction is seen to be alkaline. Indol is seldom produced by these microörganisms. They belong clearly to the "proteus vulgaris" group.

Group III. Bacillus Cloacae Type.

Bacteria of the "cloaca" type have been encountered from time to time in the tap water. They have a morphology and motility like that of *Bacillus coli* and on the surface of agar they look not unlike this microörganism. They acidify and coagulate milk, and produce non-spreading colonies in agar and gelatin. The latter medium is liquefied and the precipitated casein in the milk tubes slowly digested. The organisms ferment dextrose actively, the gas produced showing an inverted formula, $\frac{H}{CO_2} = \frac{1}{3-4}$, the carbon dioxide being in excess of the hydrogen. They may or may not produce indol and in some instances their liquefaction of gelatin is extremely slow.

Group IV. Paratyphoid or Paracolon Type.

In a few samples we have isolated species which must be called "paratyphoid" or "paracolon" bacilli. They resemble *Bacillus coli* or *Bacillus typhosus* in morphology and motility, fail to liquefy gelatin, and ferment dextrose with the usual *Bacillus coli* gas formula, $\frac{H}{CO_2} = \frac{3-4}{1}$. In milk they produce a slight but transient acidity which gives place to an alkaline reaction after the lapse of four or five days. Their cultural characters place them clearly in the intermediate group.

Group V. Liquefying Organisms like Bacillus Coli.

Under this heading we have placed a number of cultures which represent a distinct type. The organisms have the greatest superficial resemblance to *Bacillus coli* in morphology, motility and growth on agar. In the dextrose fermentation tube they produce an acidity and evolve a gas which has the regular formula $\frac{H}{CO_2} = \frac{3-4}{1}$. On potato they grow in a dirty brown layer. They may or may not produce indol. While thus having many of the characters of *Bacillus coli* they are distinguished from it by their rapid liquefaction of gelatin. The liquefaction is funnel-shaped, proceeding out from the line of growth of the bacteria and not from above downward, as in the "proteus" and "cloacae" cultures. The colonies have no tendency to spread and are thus distinguished from *Bacillus proteus vulgaris*, while by their gas formula they are differentiated from *Bacillus cloacae*. The species is a distinct one, and I have found it on a number of occasions in the city water.

Group VI. Bacillus Faecalis Alkaligenes Type.

Many of the fermentation tubes which show the presence of the various organisms above mentioned give in addition colonies which develop into a non-fermenting organism of great importance. This is Petruschky's *Bacillus faecalis alkaligenes*. It has a motility and growth on agar like that of *Bacillus typhosus*, fails to liquefy gelatin, produces an alkaline reaction in milk, and in the fermentation tube grows only in the open bulb with the production of alkali. This organism occurs frequently in the city water and may be isolated both from the fermentation tubes and from agar plates.

SIGNIFICANCE.

While the microörganisms belonging to the above-mentioned types are accompanied, in our city water, by the usual water bacteria, spore-bearers, pigmented forms, etc., the latter are in the minority. The species usually encountered are the non-pigmented, non-spore-bearing, sugar-splitting bacteria, and it is important to consider what significance must be attached to their isolation. It seems to me that but one opinion can be expressed as to these findings. They indicate a permanent and serious pollution of the supply. The presence of *Bacillus coli* regularly in one cubic centimeter quantities is recognized by all sanitarians as evidence of sewage contamination and its occasional isolation from smaller quantities, 1/10 cc., is evidence that this contamination is extensive in character. The presence of these other strains is also, I believe, not without considerable significance. In some instances, as with *Bacillus cloacae,* we are dealing with an organism which is characteristically found in sewage from which source it was first isolated by its discoverer, Jordan (1). In other cases we have organisms of the "proteus" type, which may and probably do exist in nature, but many years ago I (2) pointed out that this species is, next to *Bacillus coli,* the most commonly found species in the intestinal tract of man, and it is not impossible that its presence in the city water is due to fæcal pollution. Other organisms which have been isolated, those of the "paratyphoid" and "alkaligenes" type, are essentially intestinal in character. This is especially true of Petruschky's bacillus which is present in the alimentary canal in nearly every individual. Finally the organisms mentioned above as "liquefying organisms," like *Bacillus coli* have long been viewed with suspicion by bacteriologists. They are probably identical with the organisms described in the literature as "liquefying coli" a misnomer which Durham (3) especially has deplored. I have isolated them frequently here in Baltimore from sewage, from some of the tributaries of the city water, as well as from the tap water itself, and from polluted deep water wells. They are identical in cultural characters with a liquefying bacillus which I described from the intestines several years ago and the late Major James Carroll, of Washington, stated that these organisms were very common in various samples of water examined by him in Washington and believed to be contaminated. While their significance as an index of pollution is by no means certain, as Prescott and Winslow (4) point out in regard to intestinal bacteria which are not *Bacillus coli* yet their constant isolation from the city water, which is definitely polluted by *Bacillus coli,* is not without considerable interest.

SOURCE OF POLLUTION.

We thus see that the water drawn from the tap shows the evidence of serious pollution and since these results have been obtained now for a period of six years this pollution evidently has a permanent character. If we now ask its source it is difficult, if not impossible, to point to any one particular stream which is responsible for the contamination. My own observations relate only to the district in the immediate vicinity of the small town of Cockeysville and are concerned with three especial tributaries which empty into the Gunpowder River in this locality. These small streams are Beaver Dam Run which passes through Cockeysville, the Oregon Branch, and the Western Run. The water of these three runs is grossly polluted. This can be made out simply by an inspection of their banks where the sewage of numerous farm houses and of one or two small settlements can be seen making its way directly into the streams. As these small runs join the Gunpowder not very far above the dam at Warren the origin of the contamination of the Gunpowder water is not far to seek. The evidences of pollution which may be gained by naked eye inspection are amply substantiated by the bacteriological examination of the water collected from the various runs and of samples drawn from the Gunpowder itself. The number of organisms in the river and in the tributaries is fairly high, varying from 2,000 to 25,000 per cubic centimeter, and the fermentation tube reveals a very serious pollution. The presumptive test is frequently positive in great dilution, 1/100 or even 1/1000. The species cultivated from the agar plates and from the Smith tubes conform to the types isolated from the tap water. *Bacillus coli* is most frequent, but *Bacillus cloacae,* the liquefying organism like *Bacillus coli,* and *Bacillus faecalis alkaligines* are also found with some constancy. Some of these small streams indeed seem to carry nothing but a diluted sewage. The sedimentation in Lock Raven and in the various city reservoirs has served only to diminish the number of microörganisms. It has not altered the essential character of the bacteria, the species which survive and appear at the tap being practically identical in character with those found in the Gunpowder water above the Warren Dam before any considerable sedimentation has taken place.

REMEDY.

If our ideas as to the pollution of the Gunpowder River shall be substantiated by the work of other investigators, or if other sources of fæcal contamination shall be found, as for instance in Lake Roland and its tributaries, with the study of which Dr. W. R. Stokes has been engaged for some time, then the question naturally arises as to the means which must be adopted to remedy this evil and protect the citizens of Baltimore from the risk of water-borne infections. In my opinion the pollution of the Gunpowder River is too great and the sources of contamination too manifold to permit the adoption of hygienic measures which would be stringent enough to alter the character of the water permanently and make it safe for drinking purposes. Since the Gunpowder River offers the city the most convenient and most valuable source of supply, or at least may be used as the chief portion of our system, this water must in some way be made suitable for domestic use. There is evidently but one method of bringing this about. The water must be filtered, preferably through sand filters. Fortunately, this measure has already been adopted by the city, and it is a matter of supererogation at the present time to call attention to its necessity. Our bacteriological examina-

tions serve then more to testify to the wisdom of these patriotic officials, who for some time now have insisted upon the installation of some system of sand filtration for the purification of our water supply, than to call attention to some measure which it is impossible to carry out.

Even if it be claimed that filtration will not necessarily diminish our mortality from typhoid fever, the chief water-borne disease, we must remember that there is abundant evidence to show that the adoption of filtration plants has been followed by a diminution of the mortality from enteric fever in many cities. That this is not always the case seems to be true from the experience of the city of Washington. It may be pointed out, that this latter city forms an exception to the general rule, and that the typhoid fever was evidently not associated with a polluted water supply, but was due apparently to bad hygienic conditions in regard to the milk and food furnished the city. We believe, however, that our water supply is contaminated, and that it should be purified on general principles of good sanitation. If it diminishes the typhoid mortality, then we shall indeed be fortunate. But we do not know the source of *all* of our typhoid fever. Some may be water-borne and personally I am inclined to believe that much of it is associated with our polluted supply. But some may be milk-borne and some may be the result of a direct transmission of the typhoid bacillus from one individual to another. Any one who has examined, over any length of time, the milk sold in the little shops in the poorer sections of the city, where the bacterial count may reach 3 to 4 million to the cubic centimeter, must realize that the conditions which allow this great bacterial pollution will also allow a pollution of the milk by *Bacillus typhosus*. And any one who has read that valuable little contribution of Baetjer (5) on an epidemic of tyhoid fever in Baltimore County, where 17 cases of typhoid occurred in two houses, must feel that part of this disease in our midst is due to direct infection or to chronic bacillus carriers. Let us first provide Baltimore City with pure wholesome water and eliminate our polluted supply as a possible source of typhoid fever. We shall then be in a position to devote all of our energies to a further study of this malady, ascertain more accurately the origin of the disease and adopt those preventive measures which modern hygiene is teaching us are effective in controlling the spread of this most serious infection.

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A CASE OF CONGENITAL DEFORMITIES (PATENT DUCTUS BOTALLI; ABSENCE OF LEFT KIDNEY AND CHRONIC PERIPHERAL ŒDEMA).

By Charles R. Kingsley, M. D.,
Medical House Officer, Johns Hopkins Hospital.

(From the Medical Clinic of the Johns Hopkins Hospital and University.)

Jennie L., a white child, five months old, of Russian parentage, was admitted to the medical side of the Johns Hopkins Hospital, August 31, 1909. According to the mother, both parents had been well, with no history of tuberculosis or alcoholism on either side. The patient was the last of a family of eight children, all normal and healthy. There had been no miscarriages. The patient was born after a nine-months' pregnancy, during which the mother did her usual housework, but suffered no trauma. The presentation was breach, but the labor was easy. Soon after birth the mother noticed that the child's hands and feet were swollen and the tongue was too large for the mouth. No attention was paid to this since the child nursed well and grew, but the continuation of the deformities decided the mother to take the child to the hospital.

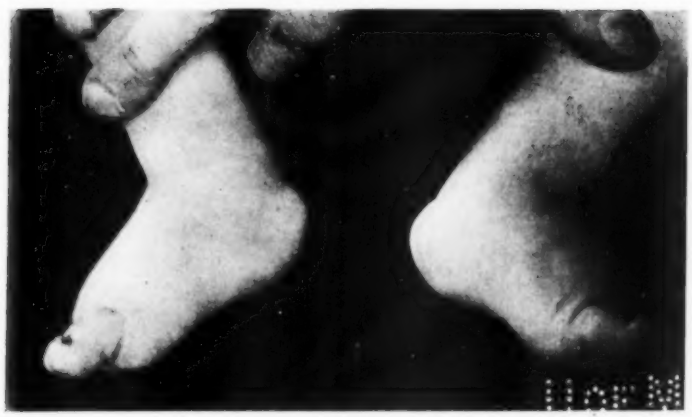
On admission to the ward the patient was found to be of average size for her age, 56 cm. long, and fairly well nourished, weighing 5675 gm. She appeared normally bright and intelligent, noticing people, surrounding objects, and moving about actively. The skin was soft and smooth, with no eruption anywhere. The head was held up fairly well and seemed normally formed, with a small anterior, and an almost entirely closed posterior, fontanelle. There was no brachycephaly with shortening of the anterio-posterior diameter and flattening of the occiput, the measurements being about normal for a child five months of age. A growth of fine black hair covered the head; the eyebrows and lashes were long. There was a peculiar bluish pallor of the face, but no cyanosis of the lips and finger tips. The neck presented an odd appearance due to two folds of redundant skin which ran from behind the ears to the shoulders, extending about 8 cm. in breadth when stretched. A slight prominence of the eyes was noticeable; ocular movements were normal with no strabismus or nystagmus. There was no epicanthus and the face was not that described as typical of a mongol. The pupils were equal, reacting normally; scleræ clear and conjunctivæ of good color. Large, thin, flabby ears protruded at right angles to the head. Hearing was absent on the left side, and a purulent

FACE: NOTE LARGE TONGUE AND EARS.

CUTANEOUS FOLDS—FROM BEHIND.

HANDS SHOWING PECULIAR FINGERS AND OEDEMA.

CUTANEOUS FOLD—SIDE VIEW.



FEET: NOTE PUFFY OEDEMA.

discharge was found in the left external meatus. Breathing was unobstructed and the nose normal looking. A huge tongue protruded through the lips to a level with the surrounding skin. There was no furrowing or enlargement of the papillæ, but the tongue was coated and indented by the upper and lower alveolar ridges. The saliva was swallowed. There was a highly vaulted palate and a large uvula but no abnormality of the tonsils and pharynx. The lips were not especially thick or protruding and there were no rhagades. No glandular enlargement was found. The thyroid could not be felt and there was no substernal dulness to suggest a thymus gland. The thorax was well formed, free from rickety rosary and Harrison's grooves. In the notch of the epigastrium the cardiac impulse was most forcible and on percussion the heart's dulness was normal in location and size. On auscultation there was an embryocardia and a sawing systolic murmur, following the first sound, well heard over the left chest. At the base the systolic murmur was especially loud and vibrating with a point of maximum intensity over the upper third of the sternum; both second sounds were clear. The pulse was regular, of good quality, and well felt in the extremities. The abdomen looked natural, without marked prominence or hernia. A soft liver edge could be felt about 2 cm. below the costal margin. The spleen was not palpable. The genitalia were normal in all respects.

The extremities were bulbous, showing considerable swelling of the lower parts of the legs and forearms. The skin looked waxy and bluish over the dorsa of the hands and feet, pitting readily on pressure, but not thickened or indurated. The extremities were cold. The fingers and toes were long, showing a fusiform enlargement of the two proximal phalanges which were in slight flexion, the distal phalanx being in slight extension. A transverse line of constriction formed a groove at the terminal phalangeal joints, most marked in the fingers, but present in the toes to a lesser degree. Both finger and toe nails were growing almost at right angles to the digits, apparently pushed up by an overgrowth of the terminal phalangeal tissue. The muscular power and sensations seemed normal.

Examination of the blood showed a red cell count of 4,900,000, white cells 10,000, and hæmoglobin 78 per cent. The urine and fæces were normal. A Wassermann test, done on the blood serum, gave a negative result. X-ray plates of the hands showed no abnormality.

The child was kept on graded doses of thyroid extract in the belief that the condition might be an atypical form of cretinism, but after two months, there being no improvement, the drug was discontinued. Small doses of diuretic drugs combined with digitalis failed to affect the œdema. The tongue remained as on admission, large and protruding. The greatest difficulty was encountered in finding a modified milk suitable to the child's delicate digestive organs. There were several gastrointestinal upsets with vomiting, diarrhœa and a temperature of 101° F. The weight remained about as on admission with occasional fluctuations. The temperature, aside from the gastric upsets, was normal or slightly below; the pulse rate was normal. In January the child seemed weak and feeble, and on the 9th of the month her temperature rose suddenly, with a profuse diarrhœa and vomiting. There was no improvement in the condition and exitus occurred next day.

Autopsy (Dr. Whipple).—The interesting points in the autopsy findings are as follows: a very inconspicuous thymus, there being practically no tissue where the gland should be. Normal heart valves, but an open ductus Botalli, measuring about one millimeter in diameter. An acute diffuse cellular myocarditis. No special hypertrophy of the ventricles. Scattered areas of broncho-pneumonia in both lungs. The left ureter appeared normal at its entrance into the bladder and as far as followed up to the brim of the pelvis. Here it was lost and no remnant of kidney tissue was found in this area. The left adrenal was close to the diaphragm and appeared normal. The right kidney was very large, about the size of two normal kidneys. There was a single distorted pelvis coming from the anterior median portion of the irregular kidney mass, the long axis being nearly parallel with that of the vertebral column and lying almost completely to the right of the vena cava, which crossed over its mesial surface. The kidney substance looked in no way abnormal. An acute enteritis throughout the ileum was found.

The thyroid was small, pale, and uniform on section. The bone marrow appeared normal. Unfortunately, no permission to examine the brain could be obtained.

TRANSVERSE FRACTURE OF THE BODY OF THE ISCHIUM IN 1893: TERMINAL DISPLACEMENT IN 1902: DIAGNOSTICATED IN 1909.

By NORVELLE WALLACE SHARPE, M. D.,
St. Louis, Mo., U. S. A.

The case herewith detailed is not classified as unique, but of extreme rarity and interest.

Fractures of the ischium, participating in a general pelvic crush, are not notably rare.

Fractures of the ischium, without coincident pelvic fractures or complications are considerably more infrequent, and are worthy of record.

Fractures of the *body* of the ischium, the result of direct impact, and without coincident pelvic fractures or complications, producing but moderate disability and remaining undiscovered for an extended period of time, are not only noteworthy on account of interest and rarity, but specially merit consideration in conditions which involve a differential diagnosis.

The following dicta from *Scudder, Keen,* and *Stimson* are recorded as suggestive:

Scudder, "Treatment of Fractures," 1910; "fractures of sacrum, coccyx, symphysis pubis and ischium are extremely rare."

Keen, "Keen's Surgery," Vol. II, 1907; "fractures of the ischium, sacrum, and coccyx . . . . are very rare."

Stimson, "Fractures and Dislocations," 1907; "fracture of the ischium is one of the rarest of fractures of the pelvis. *Malgaigne* collected only six cases and his list has not been since increased by any reported in detail. In some of the cases almost the entire ischium was broken off, in others only the tuberosity. Experiment indicates that a fracture may pass into the cotyloid cavity. In three of Malgaigne's cases the cause was a fall upon the buttocks; the fourth a gunshot fracture, the fifth caused by an explosion, the sixth due to forceps manipulation, in a case formerly with the pelvic ring fractured, and ischium was broken. In the simple cases there was little or no displacement; in the gunshot fracture the fragment was displaced downward more than 2 inches by contraction of ham-string muscles, the displacement persisted but does not appear to have interfered materially with the movements of the limb."

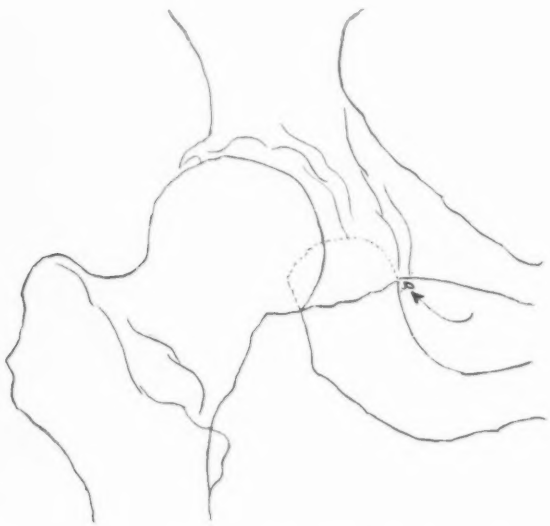
Transverse fracture of body of right ischium (about ¼ size of original plate). Note ¾″ overlap of the caudad fragment (*cf.* Section 2 of Dr. Carman's report).

H. D. B., aet. 64, has led an active mercantile life, relieved by field and water sports abundant in quantity and choice in character. The patient is vigorous and well muscled, 5 feet 5 inches in height, weight 165 pounds, he exhibits a substantial amount of adipose. For some years has "had trouble with right leg"; an attack of left "sciatica" in 1901, and more or less so-called "sciatic pains" on both sides since. Right hip and thigh became troublesome in 1903; "could not keep up in my shooting"; "friends noted a slight drag," etc. Pains now began in hip, back, knee and foot, rarely at night, but more or less constant when walking; this pain and "consciousness of having a hip" have thus existed *seven years.* Patient has consulted numerous physicians both in the States and abroad, the various diagnoses leaning to some form of sciatic involvement. In 1909 an X-ray examination was made at a New York up-state sanitarium, and a diagnosis of "trochanter split and piece off of one side," rendered.

For the aforesaid "sciatica" relief has been sought through applications of electricity, liniments, Swedish movements, massage, "light treatment," leucodescent lamps, etc., but all without avail.

The trouble, not disabling, but ever present, remained without improvement, indeed it grew worse.

His condition was brought to my attention last November (1909); before any examination was made, he was referred to Carman (St. Louis) for a searching X-ray exploration. A diagnosis of fracture of right ischium was returned. At a later consultation (April 29, 1910), an effort was made to discover an anamnesic clue that would indicate when the fracture had occurred and how it had remained so successfully undiscovered.



KEY TO PLATE.—Contrast sharp angulation at *a* (caused by cephalad displacement of caudad fragment) with normal graceful periphery of obturator foramen.

Patient's record shows that in 1897 he sustained a severe fall on a marble floor; a critical orientation of the facts coincident with and sequent to this accident leads to the conclusion that in no manner should it be considered the cause of the fracture. An antecedent accident proved more promising; in truth *the only casualty in the record that warrants serious consideration.* The details are:

In 1893, while fishing, patient sustained a severe fall, striking his right buttock on "a pointed rock" (in shape, I take it, akin to a cypress knee, familiar to those who shoot

or fish in the swamp and overflow lands of the South); there immediately followed intense local pain (patient states that he "was almost paralyzed"), this remained for several hours, but by the following morning merely the ordinary sensations of a severe contusion were present. No essential disability followed, and though moderate discomfort was more or less constantly in evidence he continued an active life, including his shooting and fishing. For this discomfort, during the succeeding years, he was subjected to the various forms of treatment above noted.

In 1902 while indulging in some "physical culture" exercises (ordered for the existing trouble), during which the right leg was extended and the thigh flexed sharply on the abdomen, *"something snapped"* within the right pelvis. This sensation was distinctly experienced by the patient, and beyond peradventure, his memory in this regard being quickened by the fact that he recalls having announced to a relative that as a result of this incident, he was "done with physical culture" and allied forms of treatment.

In 1903 hip and thigh became more troublesome, he could not maintain his former speed and endurance in a day's shoot, and his disability became patent to his friends (as noted above).

Most careful questioning fails either to shake the evidence just recorded or to introduce any other accident that might with reasonable probability be held to have definite etiologic significance.

The conclusion is therefore compelled that this ischium was fractured in 1893, and remained *undiscovered for sixteen years*, i. e., up to November, 1909.

The interesting question arises, is it not possible, during the energetic movements of the "physical culture" episode of 1902, that the overriding of the caudad fragment (see X-ray photograph) was developed; and that antedating said episode (i. e., 1893-1902) the fragments had remained in fair terminal apposition, maintained either by a reasonably intact periosteal bridge or an elastic fibrous bond, competent for all ordinary demands, but which yielded to the sudden violence of 1902 with consequent overriding. Furthermore, is it not probable that this overlapping displacement, once established in 1902 has continued, without reduction, to the present day?

It is remarkable that in a man, very intelligent, capable of giving a lucid description of facts and symptoms, anxious to maintain himself in the pink of condition, comfortably well-to-do, and under the observation of numerous physicians; it is indeed remarkable that such a fracture should remain *undiscovered for sixteen years*. There are two possible explanations: first, X-ray orientation had not been developed to its present accuracy in the earlier years of his disability, and as a result was neither frequently recommended nor often employed; second, the combination of a well-to-do patient, exceedingly solicitous regarding his physical condition, presenting a syndrome readily assignable to "sciatica" with no strikingly exceptional or highly suggestive clue leading toward an exhaustive examination and a more exact diagnosis—this combination probably proved the pit into which "all fell." It is stated that at no time during these many years was a rectal examination made.

Obviously: *Haec fabula docet rationem examinem sine cura culpam gravem esse.*

Present Condition.—Pain is moderate when seated or in bed, but constant when walking, it is accentuated during damp or unsettled weather. He uses a stick, and evidently "nurses" the right leg during locomotion, for excursion of the knee is automatically limited. States that he is liable to stumble over small obstacles; the resulting "jars" are painful, radiating down the thigh. Can definitely identify (and claims to have always been able to definitely identify) a relatively sensitive area, i. e., the tuberosity of the right ischium and peri-ischial structures. He continues to engage in a fairly active life but, handicapped as above, has eliminated the more vigorous forms of his customary sports.

Comparative measurements show relative dextral symptosis:

	Right.	Left.
Circumference calf	14″	14¼″
Circumference thigh lower ⅓	15¾″	17″
Circumference thigh middle ⅓	20⅛″	21¼″
Mid. symphysis to postmesial line	18″	21″
Ant. Sup. Spine Ilium to Internal Malleolus.	31½″	31¾″

The mid-symphysis postmesial measurement is particularly interesting in that it records dextral gluteal change.

Rectal examination reveals an area of the right ischium sensitive to pressure. This area exhibits a moderate entad boss, which accurately corresponds to the overlap of the caudad fragment of the ischium (see X-ray photograph).

Dr. Carman, to whom full credit must be accorded for supplying the correct X-ray diagnosis, in his report calls attention to—

1. A spear-like exostosis springing from the superior edge of the acetabulum; furthermore the articulation outline is indistinct.

2. There is fully a ¾-inch overlap (original plate practically life-size); the caudad fragment seen partly through the acetabulum, partly through the femoral head, being shown mesad to that extent.

3. If patient had not been so stout, a series of perspective views might have been obtained that would have afforded data regarding character of displacement and union.

Based upon Section 1 of Carman's report it would seem to be evident that a moderate hypertrophic arthritis is present. It is entirely conceivable that a portion of the gluteo-femoral syndrome is due to exacerbations of this condition.

Technically, it is entirely possible that pressure pains might be largely, if not wholly, relieved in such a condition by an attack directed toward the overlapping fragments.

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PERI-TONSILLAR ABSCESS.

By Mactier Warfield, M. D.,
Instructor in Laryngology, Johns Hopkins University.

This abscess is always painful, and at times dangerous. The laryngologist rarely sees one outside of a hospital, for the great majority of them come to the general practitioner, who either treats them after the method generally given in the text-books, a method often unsuccessful, and always dangerous, or frightened by its dangers and mindful of past failures, bears with resignation the sufferings of his patient until the abscess opens of itself. My excuse for this paper is that I wish to present a method of opening these abscesses, one which I have used for some time, that has never failed me, and which I believe to be perfectly safe, hoping that others may try it, in order that we may arrive at a correct judgment as to its value.

By a peri-tonsillar abscess, I mean one which is the result of an infection of the loose connective tissue immediately about the tonsil which presents the symptoms with which we are familiar as belonging to quinsy. Since it is supposed that the starting point of the infection may be anywhere in the peri-tonsillar connective tissue, the resulting abscess has no definite position where we may always look to find it. As it is also accompanied by great swelling and œdema of the mucous membrane over it, the area over which we can get fluctuations is therefore increased, and as the mouth can barely be opened, and hardly anything can be seen of the throat, it becomes impossible to definitely locate the abscess and find its point of greatest fluctuation. Examination by the finger affords the most accurate information, but this gives so much pain that few patients will submit to it.

In opening an abscess the procedure usually given in the text-books is this: when in any particular case an incision is decided upon, it is not attempted, as a rule, to definitely locate the abscess, but the incision is made at a site which experience has shown to be the usual point of greatest swelling, and where pus can be found. This incision is begun at a point opposite the junction of the uvula with the soft palate and about three-eighths of an inch external to the inner border of the anterior pillar of the fauces, and is carried from above downwards about one-third the distance from the point of origin to the lower border of the tonsil. The knife is wrapped to within a half inch of the point, and is plunged into the swelling to the depth of half an inch, care being taken not to wound the tongue on withdrawing it. Often when there have been unmistakable evidences of the presence of pus, and when the incision has been carefully made, the abscess is not opened.

There is hardly anything which occasions the physician greater chagrin than a failure of this kind, or does more to lower his reputation with his patient, who has had his pain increased instead of receiving the promised relief from suffering, and who now considers the doctor the cause of all his troubles. This incision is not without danger. In making it the ascending pharyngeal artery has been cut and the carotid also. Bosworth says that he never opens a peri-tonsillar abscess without a certain feeling of nervousness on that account.

To permit the abscess to open spontaneously has also its dangers. Abscesses have burst in sleep, and the pus entering the air-passages has drowned the patient. Pus has burrowed down, and found a way out in the axilla, or entering the pleural cavity has caused death. Most abscesses, when let alone, open of themselves between the fifth and tenth day, but at times this does not happen for five or six weeks, and by that time the patient is reduced to a pitiable condition from pain and want of food and sleep. Sometimes, either through fear of accident, or because of former failures, the patient or his family will not allow the abscess to be opened.

In a long service in the throat clinic of the Johns Hopkins Hospital, it has been possible for me to see a large number of peri-tonsillar abscesses, and to make observations which have led me to hold opinions as to their origin, and the best way of opening them, which differ from those usually held.

In the first place, instead of considering that the infection may enter, and the abscess arise at any point in the peri-tonsillar connective tissue, it seems quite evident that it always starts from the same point, and that point is in the supra-tonsillar fossa, and that the abscess is always the result of the extension of the infection to a cavity which opens into this fossa. This cavity or space is in the connective tissue, and extends outward along the superior border of the tonsil, and downward along its outer border, thus bounding it on two sides. The abscess begins at the supra-tonsillar fossa, and as pus continues to be formed it occupies first that part of the cavity which is above the tonsil and then the part on its outer border. As the part on the outer border of the tonsil becomes distended with pus, it forces the tonsil out of its usual position, and pushes it inward over towards the uvula, making it appear as if it were swollen itself, but its increase in size is only apparent and not real, for the abscess is never within the tonsil, but external to it.

The chief causes of peri-tonsillar abscesses are acute follicular tonsillitis, imperfectly removed tonsils and rheumatism. After an attack of acute tonsillitis, the pain and fever usually disappear at the end of three or four days, and the patient thinks he is getting well. But it is often only the lull before the storm. At the end of twenty-four or thirty-six hours, he feels a sudden sharp, stabbing pain in one tonsil, and that is the beginning of the abscess. The swollen tonsil has carried an inflamed crypt into the supra-tonsillar fossa, and infected it. Or the fossa may be infected by the small chronic abscesses which are the result of leaving parts of tonsillar crypts behind in removing a tonsil.

The abscess which is the result of the acute rheumatic

infection, starts as a small, circumscribed, deeply inflamed area over the supra-tonsillar fossa and the adjacent anterior pillar. It can be aborted if seen very early by the use of salicylate of soda.

Whatever the cause of the abscess may be and whether it is large or small, it can always be located in the supra-tonsillar fossa, which itself can easily be found even where the throat is very much swollen and distorted. This then is the place where the abscess is to be opened in every case.

The necessary incision is made with a curved bistoury, which is held parallel to the soft palate, with the edge of the blade looking forward, the back towards the posterior pillar, and the point directed outward towards the ear. The point is entered in the supra-tonsillar fossa, in the angle between the pillars of the fauces as high up as possible, and is passed outward until it meets with an elastic resistance, which is the abscess. This is entered, and the knife is brought out straight forward through the anterior pillar. As a rule the knife does not have to enter more than a half inch to reach the abscess. The cut in the anterior pillar is through swollen mucous membrane and does no harm. A bent probe can then be passed through the opening made in the abscess, over the tonsil and to its outer side, thus demonstrating the existence of the cavity there. As the ascending pharyngeal artery and the carotid are at the back of the knife, they are in no danger of being cut.

This incision, then, has these advantages: it is perfectly safe, it is easily made, and it is always successful.

SUMMARIES OR TITLES OF PAPERS BY MEMBERS OF THE HOSPITAL OR MEDICAL SCHOOL STAFF APPEARING ELSEWHERE THAN IN THE BULLETIN.

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NOTES ON NEW BOOKS.

Symptomatic and Regional Therapeutics. By GEORGE HOWARD HOXIE, A. M., M. D. (*New York and London: D. Appleton & Co., 1910.*)

The title of this work excites curiosity as to its exact scope for as a rule it is not wise for too much attention to be directed to the treatment of symptoms. The temptation—too frequently succumbed to—is to give care rather to the relief of symptoms than to getting at their cause. Obviously in some cases we cannot do anything more than treat symptoms, as in hopeless cases of malignant diseases. In some cases special attention to a particular symptom is important and such a section as is here given on the treatment of itching is excellent, but to treat emaciation as a symptom hardly seems very good therapeutics. The reasons are not evident for placing some conditions under certain headings. Thus it is difficult to see why the serum treatment of diphtheria should be discussed under the heading of "chronic inflammations." Even "regional therapeutics" can hardly justify the placing of typhoid fever in the section dealing with diseases of the digestive tract.

For the consideration of a particular disease the discussion of the treatment of typhoid fever was chosen. There are some points which deserve notice, thus the author says that "it should be remembered that the stools must be green and inoffensive. . . ." Now green stools do occur in typhoid fever, but this is rare and it is hard to understand why they must occur. The direction that "there must be no gas pains" seems rather a curious one. The author does not advocate the bath treatment, but this is not surprising when we read his description of a tub bath (p. 28) in which he says "In giving the bath, one should be careful to keep the patient in the water only a minute or two. . . ." It would be interesting to know the authority for this statement; the reviewer knows of none. It is the custom of many writers to pass criticism on the bath treatment of typhoid fever when it is evident that they do not know the method of carrying it out.

There are some descriptions of treatment which do not seem very rational. Thus in endocarditis with valvular insufficiency it is advised "to strengthen the valves by very carefully graduated exercises and passive movements (Schott or Nauheim treatment)." That the object of this or any other treatment is to strengthen the heart valves is a new point in therapy. Under pneumonia it is advised to give atropine to dilate the capillaries and equalize the blood pressure when the skin becomes clammy and pale. This last phrase is not quite clear but as regards any need of *dilating* the capillaries, the usual opinion is that when the circulation is affected in pneumonia, there is too much vasodilation. Then there is a generally accepted opinion that atropine tends to contract the peripheral vessels. Angina pectoris is discussed under the heading of cardiac neuroses and the treatment here given would not carry one far. There does not seem to be any mention of aneurism at all. Broncho-pneumonia is discussed under the heading bronchitis. This perhaps is regional therapy, but it does not seem likely to aid in obtaining a broad view of therapeutics. In gastric hyperacidity a dose of soda of one to two grains is advised, but it is difficult to share the author's view that this will be efficient. In peritonitis "distressing vomiting and peristaltic frenzy are kept in abeyance by careful gastric lavage. The term "peristaltic frenzy" sounds well, but are the bowels not apt to show absence of peristalsis in peritonitis when the stage of vomiting is reached. In several places the author condemns certain methods of treatment, but still devotes space to describing them (for example, the use of gelatine injections on page 275).

It may be argued that a complete discussion of treatment should not be expected in a work with this title but if so there seems little reason for its appearance. The extracts quoted are enough to suggest doubt as to the value of the work and we cannot recommend it. The whole idea of the book seems a step backward.

Nephrocoloptosis. By H. W. LONGYEAR, M. D., etc. (*St. Louis: C. V. Mosby Company, 1910.*)

As the title suggests this interesting little volume is an exposition of a relationship supposed to exist between nephroptosis and coloptosis. The interest centers about the nephrocolic ligament, a structure which the author claims to have discovered. It is described as consisting of a number of fasciculi of fibrous tissue, which can be anatomically grouped and easily demonstrated as thus forming a definite band or ligament of considerable tensile strength extending between the right kidney and the ascending colon, and between the descending colon and the left kidney. The fibrous network of this ligament is continuous with that of the fatty capsule of the kidney.

Nephroptosis without coloptosis, it is boldly asserted, does not occur. In fact the prolapsed colon, weighted down with accumulated fecal masses as a result of associated constipation, and tugging on the right kidney by means of the nephrocolic ligament is considered the principal contributory etiological factor in movable kidney.

Acting upon this idea the author has devised an operation for the correction of nephroptosis the essential feature of which is the utilization of the nephrocolic ligament as a substantial supporting structure.

There are chapters dealing with the subject of movable kidney from the standpoints of Anatomy and Pathology, Etiology, Symptomatology, Diagnosis and Treatment; and at the end is appended a somewhat detailed report of 54 cases of nephrocolopexy in substantiation of the author's claims.

The illustrations are numerous, clear and helpful; and the publishers' work is creditably done.

The book will doubtless stimulate surgeons generally to search very carefully for the nephrocolic ligament in all cases operated upon for nephroptosis. It seems very strange that such a definite anatomical structure, of such vital importance etiologically in a

relatively common disorder, as described and pictured by Dr. Longyear should have escaped notice so long. But judgment as to its real worth must be withheld, of course, until the author's claims have been thoroughly tested by other surgeons. Any definite contribution to the study of movable kidney from an etiological standpoint should be gratefully received, and justly merits careful consideration, in view of the present chaotic state of our knowledge upon this subject. For one cannot help being impressed by the curious discrepancies and radical differences of opinion met with when comparison is made between the views of different authors who have made a special study of movable kidney. Thus, for example, F. W. Griffith (Johns Hopkins Hosp. Bull., XXI, 75) who made a very elaborate and painstaking study of body-shape in relation to movable kidney, concluded that faulty body-shape is more important as an etiological factor than all others combined.

Not so Longyear, however, who holds this to be only one of many minor contributory causes, regarding chronic constipation in its relation to coloptosis as of very much more importance. Thus he explains the much greater frequency of right-sided nephroptosis, since fecal matter does not accumulate in the descending colon; and his statistics show that of 155 cases of movable kidney found among 1000 women examined, he met with not a single case occurring on the left side alone.

Griffith, on the other hand, reports from a study of both clinical and operative statistics at the Johns Hopkins Hospital that the correct proportion of right- to left-sided nephroptosis is about three to one! He found furthermore that a history of constipation was obtained in 49 per cent of white women and 70 per cent of colored women; or, as he says, "White women in whom movable kidney is common do not suffer from constipation as frequently as do colored women in whom movable kidney is uncommon."

It is evident, therefore, how far we are from any uniformity of opinion upon this subject at present, and Dr. Longyear is to be congratulated on having contributed in this attractive little volume so much food for reflection, which it is safe to predict will stimulate the surgical world to speedily interest itself in a thorough test of his original suggestion. E. H. RICHARDSON.

Andreas Vesalius, The Reformer of Anatomy. By JAMES MOORES BALL, M. D., St. Louis. Quarto size (10 x 12¾ inches), printed on heavy Normandy vellum, deckle edge, with heavy extension cover to match. A de luxe volume, issued in the highest style of the typographic art as regards page format, type cutting, decoration and solidity of ink. Pages XVII + 149. Fully illustrated. Price $5.00. (*St. Louis: Medical Science Press, MDCCCCX.*)

There has long been need of a publication in English giving, in readable form, the available data in regard to Vesalius. Burggraeve's Mémoire (Gand, 1841) and Roth's scholarly biography (Berlin, 1892) have largely supplied this want for a French and German reading profession. It has been a regret to all, interested in Medical History, that the promised "Vesalius" in the Masters of Medicine series has never appeared, for what is wanted is such a life of Vesal as Paget gave us of John Hunter and Power of William Harvey in this series. Doubtless Dr. Taylor found it a large task, and in view of Professor Roth's exhaustive study covering many years of labor, there must have been hesitation to enter the same field. However, there may always be something new for the subsequent biographer—*vide* the long series of Harvian orations—and Roth himself has shown, by a number of later papers, that the last word on Vesalius was by no means said in his comprehensive volume.

There are many points of view from which a biographical sketch may be written, and Roth's much annotated "Andreas Vesalius" would, if translated, probably make rather dry and uninteresting reading for the average physician. Few combine the scholar's knowledge with a style suited to all readers.

Though Dr. Ball makes no pretense of having made original studies, his narrative will give to English readers, unfamiliar with Roth and Choulant, a pleasant and helpful picture of Vesal, his contemporaries and his times.

For its interesting introductory chapters and for the review of anatomy in ancient times, it is to be hoped that the volume will be largely read, but as a picture of Vesalius it falls short of one's expectations. The narrative is broken up by the insertion of fragmentary and isolated biographies of Vesal's forerunners and contemporaries to the distraction of the reader's attention from the main theme. To really do justice to the subject would require the pen of a Henry Morley and a knowledge of the 16th century equal to his, and one could wish that he had added a Vesalius to his Jerome Cardan and his Palissy the Potter. His short sketch in Fraser's Magazine in 1853 is an indication of what might have been expected, and he did us a service—to which Dr. Ball refers—in calling Vesal the Luther of Anatomy.

Not only in its content, but typographically, the volume has much deserving of commendation. The Medical Science Press is to be congratulated on the appearance of the work, and the proofreader, on the scarcity of typographical errors. An "of" is missing from line thirteen, page 76, etc. The book is printed on heavy paper, Normandy vellum, imported from France—a commentary on the protective tariff—which takes the type and blocks of the numerous illustrations as no American made paper can. Unfortunately it has been necessary to use two qualities (pages 81-96 in the copy used for review). The index would seem to be unnecessarily elaborate—a rare and perhaps a good fault.

The attention of medical students should be directed to this volume, though it is rather too expensive a work for the majority of them to hope to possess. H. C.

Urinary Surgery—A Review. By F. S. KIDD, M. B., B. C. (Cantab.), F. R. C. S. (*London: Longmans, Green & Co., 1910.*)

In the preface the author states that his purpose is to supply practitioners of medicine with a practical treatise which will aid them in becoming more familiar with the diseases of the urinary system.

The book, of 412 pages, is divided into five sections: 1. Diseases of the Kidney; 2. Diseases of the Ureters; 3. Diseases of the Bladder; 4. Diseases of the Prostate; and 5. Diseases of the Urethra.

English medical books are usually carefully written; this one, however, is a striking exception, for the English shows a violation of most of the rules of rhetoric and a careless use of those of grammar. Ideas of slight relation to one another are connected by conjunctions and jumbled together in the same sentence; prepositions are misued, verbs are omitted, inelegant expressions are injected here and there, and words are often used which inaccurately express the meaning intended. There is almost an entire lack of coordination and unity in the long sentences and many of them require a second or third reading before the author's meaning is apparent.

A few examples of the author's English construction may be quoted: "The patient should lie up in bed, with the foot-end slightly raised, either on her back, or on her right side, and the attack will pass off in a few hours." "Stone is the commonest cause of single cyst in the kidney met with at operation." Referring presumably to the X-rays which were spoken of in a preceding sentence, he says, "Before their advent stones were approached in the dark." "Intervals of years may intervene." "The symptoms cool down." "The abscess breaks on to the skin." "A triangular shadow in the renal region with a nose

pointing downwards with phosphatic urine means ureteric block, ..." and "gross parasites such as hydatid cysts."

The arrangement of the subject matter in short paragraphs with distinctive headings is faulty and in many instances very confusing.

It does not seem necessary in a work of this kind to give the embryology of the kidney and ureter; if the reader does not know it beforehand, he will not be much enlightened after having read the synopsis.

In what should be a very important chapter on renal calculus, there is nothing new, and the arrangement of the facts given is so poor that a clear idea of the subject cannot be obtained.

The discussion of new growths of the kidney is very inadequate, in many places being reduced to simply a naming of the conditions. Inflammations of the bladder and prostate are somewhat better handled, but here again the author falls short.

The pathological descriptions in various chapters for the most part show crudity and unfamiliarity with this part of the subject. Expressions such as "The pathology of spread," "the giant cell systems," "degeneration of the higher cells of the kidney" seem to be without warrant.

In a book intended for practitioners symptomatology is the most important feature, but the signs and symptoms are divided into so many sub-headings and so scattered in various paragraphs and in general so inadequately dealt with that a comprehensive grasp of them is rendered impossible.

In the section on tuberculosis of the kidney in speaking of catheterization of the ureter, the following astounding assertion is made: "Before removal, half an ounce of a one per cent solution of corrosive sublimate is injected through the catheter." This surely would kill all the tubercle bacilli which might have been artificially introduced, but at the same time, it would destroy the ureter and pelvis of the kidney. Another statement in the chapter on Tuberculosis of the Bladder, while not dangerous, is certainly erroneous: "If a young man without venereal taint who usually sleeps well is constantly awakened at night by desire to micturate, the diagnosis is tuberculosis of the bladder."

The treatment of certain diseased conditions is short and in many instances too cursory to be of much help. The operative technique is so briefly described that it is questionable whether any mention of it need have been made.

There is much good material in the book; the author's advice for the most part is sound, his insistence on a thorough examination at the onset of various maladies is commendable, and the surgical principles which he sets forth are, with a few exceptions, correct. If the whole text were entirely rewritten in passable English, the subject matter rearranged, the errors corrected and numerous repetitions omitted the book would have some value; but as it stands, it is at best of doubtful utility.

The Sexual Disabilities of Man, and Their Treatment. By Arthur Cooper, Consulting Surgeon to the Westminster General Dispensary, etc. Second edition. Revised and enlarged. Price $2. (*New York: Paul B. Hoeber, 1910.*)

The book is divided into two sections: (1) Sterility and (2) Impotence.

In the first, the author discusses the normal and abnormal characteristics of the seminal fluid, the changes found in the spermatozoa, the various physical defects, and the most common diseases which are held responsible for sterility. At the end treatment is advised for the most usual and important changes found.

Impotence is dealt with under two headings: Secondary and Primary. (1) Secondary impotence is caused by definite morbid conditions such as physical defects and local and general disease. (2) Primary impotence is mainly nervous in origin and only slight, if any, local changes are demonstrable. Three main factors are discussed: (1) The psychical element; (2) The effect of abnormal seminal emissions; and (3) The result of irritation or over-stimulation. At the end a fairly comprehensive treatment is given.

The book is well and pleasantly written and covers the ground in a fairly satisfactory manner, but in a few places the author has been too brief, and in others, matter has been inserted which might well have been left out. The practitioner or student who knows very little of the subject may find the work helpful, but for those who are conversant with the literature it will have almost no value.

Surgical After-Treatment. By L. R. G. Crandon, A. M., M. D., Assistant in Surgery at Harvard Medical School. Illustrations. Price $6. (*Philadelphia and London: W. B. Saunders Company, 1910.*)

There are very few surgeons who have not at one time or another, in the course of their career, felt the need of a good book on "Surgical After-Treatment." Especially helpful must such a work be to the surgical beginners.

This long felt want is now beautifully supplied by Dr. Crandon, who is to be congratulated on the success of his book.

Starting out with a chapter on the Sick Room, Nurses' Chart, Posture, other chapters follow telling in clear, concise language of the care of the patient before and after operation, the anæsthesia, feeding, care of the wound, complications, etc.

Further on in the book the various operations are taken up one by one and dealt with specifically, such as for instance the Radical Cure of Hernia, Strangulated Hernia, Curettage for Abortion and Miscarriage, etc.

The last two hundred pages of the book are devoted to "Therapeutic Immunization and Vaccine Therapy," written by Dr. George P. Sanborn, also of Boston. This is a wise addition to the work as every modern surgeon must be familiar with this growing branch of medicine.

But, perhaps the most commendable feature of the whole book is the fact that it is distinctly not a narrow and detailed account of the methods and procedures of one hospital or one institution, but rather a sort of condensation, a résumé of the best methods in use at the present day in the various hospitals, both of this country and of Europe. One has only to glance at the numerous and well-chosen references to the literature to appreciate the truth of this statement. Bertram M. Bernheim.

Practical Pathology. By J. Sims Woodhead, M. A., M. D., LL. D., Fellow of the Royal Society of Edinburgh; Professor of Pathology in the University of Cambridge. Illustrated. Fourth Edition. Price $11. (*London: Oxford University Press, 1910.*)

The welcome accorded the previous editions of Prof. Woodhead's book is possibly the best indication of the need of a text of this type. As so much time has elapsed since the appearance of the third edition, it will be well to briefly review the scope of the volume.

The first chapter deals with a method of performing post mortem examinations, the requisites necessary, etc. The only criticism one can offer is whether it is really of advantage to devote much space to a subject which can only be learned by observation or by actual experience.

The second chapter deals with pathological technique. By the time he reaches his course in pathology the student should be familiar with the microscope. On the other hand his knowledge of the examination of fresh tissue is limited and this portion of the chapter is more satisfactory. The advantages of the study of fresh tissues and injected organs have been neglected to a great extent with the extensive use of microtomes and improved stain-

ing methods, and it is therefore the more important to emphasize the study of fresh tissue as the author does. This is followed by a rather complete description of hardening, embedding and staining methods which is doubly valuable since each method is numbered and these numbers are used continuously through the text as guides for the best methods available in the study of specific lesions. This reference system simplifies the student's work greatly inasmuch as it puts a definite mode of procedure at his disposal for most cases; it is possible, however, that just this precision may be a disadvantage since it will not necessitate any discriminating thought on the part of the student.

The third chapter on inflammation begins in the only logical manner, *i. e.*, the description of cells found in the blood and in inflammation. The description is objective with only sufficient theory to impress the unsolved cell genesis problems. The remainder of this chapter deals with inflammation and repair as it occurs in various characteristic tissues. The author's method in this as well as in the description of morbid processes in the subsequent chapters is to be strongly commended. First the gross lesion is described, then after the most appropriate manner of hardening and staining the tissue is given, the microscopic examination under definite low and high magnification is detailed. Many of these latter descriptions are more valuable on account of the excellent illustrations. In some instances these are diagrammatic and possibly more valuable for this reason.

The book is unique, its scope is different from every other treatise on pathology and is no doubt just the thing most needed by the student since it puts at his disposal ready methods for the study and interpretation of the usual lesions of pathological anatomy.

The price of this book, however, precludes any very extensive use.

Scientific Memoirs—New Series No. 37. By Officers of the Medical and Sanitary Departments of the Government of India.

Investigations on Bengal Jail Dietaries with Some Observations on the Influence of Dietary on the Physical Development and Well-Being of the People of Bengal. By CAPTAIN D. MCCAY, M. B., etc. Issued under the authority of the Government of India by the Sanitary Commissioner with the Government of India, Simla. Price 4/3. (*Calcutta: Superintendent Government Printing, India, 1910.*)

A very long and thorough report divided into 2 parts, preceded by an Introduction. Part I is divided into 5 chapters: 1, The Food-Stuffs of Bengal Jail Dietaries; 2, The Nutritive Value of Bengal Jail Dietaries; 3, Report on Six Months' Use of Special Diets in Puri Jail; 4, The Effects of the Large Quantity of Salt Given in Bengal Jail Dietaries; and 5, Some Side-Issues of the Investigation. Part II discusses "The Relationship of Food to Physical Development." The special nature of this report makes it one of interest only to the physiologist, or a few students especially devoted to problems of metabolism; such men will find it well worth a careful study.

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